ESSENTIALS OF FEVERS

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BY

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Hypertox[illegible] D[illegible]—Note the [illegible] gland with p[illegible]—[illegible] k N[illegible] pillo[illegible] and the san[illegible] na al [illegible] ge Th[illegible] be pal r

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PREFACE TO THE SECOND EDITION

THE appearance of the second edition has been delayed to some extent by factors outside my control but largely by my own preoccupation with other work As a result the book has been out of print for some considerable time and to those who may thereby have been inconvenienced I tender my apologies

I have yielded to my critics in two respects In the first place the book is bulkier by the inclusion of new sections on Typhus Glandular Fever Undulant Fever Influenza and Encephalitis Lethargica as well as by notes on Sore Throat and Food Poisoning while the article on Dysentery has been rewritten and expanded New matter has thrust its way in under most if not all the original headings for much additional knowledge has come our way since the first edition appeared As a slight compensation the introductory matter has been pruned but on balance the volume has gained appreciably in size—a hypertrophy to be regretted from the student's point of view

My second surrender has been in respect of the colour plates—an innovation on which I have not embarked without misgivings For it is no easy matter to present a faithful impression of a full scale rash within the compass of a few square inches and this would hold good even if colour reproduction were an exact and simple process which it certainly is not As I have had to be my own artist I have tried to establish a balance between the realistic and the

diagrammatic approaches, so as to give the student as clear and yet as typical a picture as I could contrive. In some cases this has led inevitably to exaggeration as for example in the punctations of the scarlet fever rash but I trust that on the whole the compromise has not proved unhappy.

The book however retains its original character which is to say that its title is meant to be taken literally. Nothing has been included that in my opinion is not essential to a proper appreciation of the subject. This austerity has naturally led to the suggestion that it does not contain enough for the student but he may make his mind easy on that score. While I have concentrated on the practical aspect there is more than enough theory to meet his needs.

Once again I wish to record my indebtedness to Dr J C McEntee for placing his profound knowledge of the subject so freely at my disposal and for his generous help with the proofs, to Dr Ian Taylor for many shrewd criticisms and helpful suggestions and to the London County Council as represented by Sir Allen Daley and Dr J F C McCartney for the use of the excellent collection of photographs at the Southern Group Laboratory. Lastly I would thank my publishers for their courtesy and their truly phenomenal patience.

C E B

LONDON *October*
1947

PREFACE TO THE FIRST EDITION

THIS little book is an attempt to compress the essentials of those acute infectious diseases commonly known as fevers into a volume of pocket size The term essentials has been liberally interpreted to include not only such theory as is indispensable but such practical details of treatment management and prophylaxis as will it is hoped prove of service alike to the student and to the young practitioner in his difficulties Sections on preventive nursing and isolation elementary epidemiology and the law as to infectious disease have therefore been included and wherever practicable a note on preventive inoculation and the control of outbreaks has been appended to the description of each condition Naturally within such a brief compass certain omissions and abbreviations have proved inevitable but they are I hope nowhere vital If the student has mastered the contents of this volume he will embark on practice by no means badly equipped indeed experience suggests that he will know considerably more than most

That practical knowledge of fevers is neither as wide nor as general as might be desired will I think be conceded by anyone in a position to judge Indeed for many years it has disputed the palm for what might be termed the cinderellaship of medicine only with mental disease ignorance of either being regarded as somehow excusable This is astonishing It is almost as if a congress of anatomists should decide tacitly to ignore the liver Put in this way the absurdity of the

situation becomes immediately manifest what is *still more surprising is that such an attitude would* almost seem to have official sanction For there is no examination of any sort in fevers not even a test of competence carried out by the teacher Now however conscientious the student he can hardly be expected to encumber his memory—already strained to break *ing point—with matter most unlikely to be asked He* is faced moreover with the astonishing anomaly that whereas he may be required (at his peril) to identify some recondite museum specimen of purely pedantic importance he will never be required to identify a case of hypertoxic diphtheria on the timely recognition of which a life and his own reputation as a physician may well depend In the circumstances is it surprising that we must often say of his management of fevers in general as Dr Johnson said of the woman preaching It is not well done Sir but the wonder is that it should be done at all ?

The student will do well to take his course in fevers seriously since *in them* the study of the phenomena of infection has been most actively and fruitfully pursued moreover the field of recognized infection is expanding daily Besides to descend to baser motives it will repay him handsomely The practitioner who blunders with children is seldom forgiven moreover his mistakes are palpable and prone to advertise themselves not to mention that it may spare him the humiliation of conceding that an experienced parent may prove a better diagnostician than himself And no book be it emphasized can replace first hand experience in the wards

It is a pleasant task to record my indebtedness to

the many friends who have assisted me in one way or another especially to my superintendent Dr J A Armstrong and to Dr J C McEntee both of whom have most generously accorded their criticisms suggestions and advice together with valuable practical assistance with the manuscript and proofs To Dr A D Allison and Dr Ian Taylor I am indebted for help on special points and to the publishers Messrs Livingstone for their unfailing helpfulness and courtesy Finally I wish formally to exclude the London County Council from any responsibility whatsoever for the procedures advocated or the views expressed on any subjects of controversy

G E B

LONDON *April* 1939

SECTION I

GENERAL FEATURES OF INFECTION

THE term fevers though traditional is misleading since it would seem to imply that all acute infectious diseases are necessarily febrile But fever though undoubtedly one of the commonest bodily reactions to infection is by no means invariable—witness diphtheria and whooping cough for example There is the further implication that since Isolation Hospitals exist for the reception of fever cases those diseases normally dealt with by them are somehow of a different nature and of a much higher order of infectivity than those which are not This again is not the case Influenza and the common cold two of the most readily communicable and be it added potentially dangerous diseases known to medicine are not usually segregated at all Again streptococcal sore throats which differ from scarlet fever merely in the absence of an erythrogenic fraction in the toxin of the organism involved and therefore present no rash are regarded with equanimity It is clear that we are not very logical in these matters

NATURE OF INFECTION

Perhaps we had better begin by recalling a few of those elementary facts of bacteriology with which we are all familiar in a general way but have not hitherto

considered from the purely practical standpoint All communicable diseases are due to the implantation and proliferation of bacteria or viruses Bacteria are living vegetable cells, indispensable in nature a small percentage of which during the course of evolution have abandoned their native habitat the soil in favour of a parasitic (if precarious) existence on man and other of the higher forms of life In general they consist of a *soma* or body of protoplasm surrounded by a protective capsule of a different chemical (and consequently antigenic) constitution These capsules are porous allowing dissolved substances to diffuse through them To subsist, and especially to multiply organisms first of all require food This they obtain by liberating ferments (*cytolysins*) which break down the cells of whatever part of the body they have lodged in From these broken down cells the organisms select such products as they require, and after digestion excrete the residue These excreta and products of their economy are often poisonous and with their secretions are known as *exotoxins* as distinct from *endotoxins* which are contained within the bodies of the bacteria and are consequently released only when the latter die and disintegrate

In addition to food bacteria require moisture a suitable temperature an appropriate hydrogen ion concentration and (usually) oxygen These are important practical points While bacteria will not proliferate on a painted wall or a pane of glass where they are deprived of food and exposed to the drying action of the air they may survive for surprisingly long periods in the most unpromising environment Some, such as the *M tuberculosum* are notorious in

this respect but even the cocci have been shown to possess much more vitality than was generally suspected and to retain their virulence in dust for many hours if not for days On the other hand direct sunlight is a powerful germicide Even certain types of virus such as the virus of smallpox exposed to the sun probably becomes innocuous but if its moisture has been conserved in say a bale of cotton and the temperature be suitable it may survive a voyage from Egypt to Lancashire Droplets expelled into the open air may be disregarded but coughed into the face of a fellow passenger in a train or into a pail of milk may initiate an epidemic

Boiling will kill all organisms but unless prolonged not those in spore form Freezing though it may inactivate and devitalize does not usually kill Infected milk therefore if boiled is safe but ice cream made from the same milk is not The really high temperatures provided by superheated steam or the hot air oven are the most effective of all bactericidal agents and are especially serviceable in disinfecting bedclothes mattresses laboratory ware and the like Certain chemicals in solution such as carbolic lysol etc which poison the protoplasm of the organism are also effective and are widely employed where heat cannot be used but they tend to be inactivated in the presence of organic matter such as pus Manipulation of the pH or the oxygen concentration is not as yet practicable on a large scale but the lethal effect of sunlight on most pathogenic bacteria is often forgotten

The predominant agents in those acute infections with which we are concerned are the *viruses cocci* and

bacilli Viruses bulk largely in our field since they are believed to cause smallpox chickenpox vaccinia herpes febrilis zoster measles mumps rubella poliomyelitis, influenza the common cold typhus * and encephalitis lethargica Virus diseases have certain characteristics useful to remember (*a*) they are the most infectious of all communicable diseases (*b*) they usually have long incubation periods (*c*) one attack usually confers a lifelong immunity (shining exceptions to the last two are influenza and the common cold) (*d*) they mostly evoke a lymphocytic response in the blood (*e*) they are often character ized by prodromal rashes (*f*) an encephalitis may follow any of them (*g*) apart from smallpox the infectivity of a given case diminishes rapidly (*h*) excluding poliomyelitis and possibly encephalitis lethargica the carrier state rarely occurs and then is probably only transient (*i*) the local lesion is almost always in the naso pharynx and often slight (*j*) while mostly non fatal in themselves complications are often lethal because the virus paves the way for other and more dangerous organisms Indeed the role of the virus in measles influenza and the common cold wherein it deprives the respiratory mucosa of its normal resistance to the streptococcus pneumo coccus and Pfeiffer s bacillus and thus permits these last to proliferate often disastrously may be aptly compared to that of the small boy who crawls through a gap in a fence with a view to opening the gate for his fellow conspirators Finally (*k*) although many viruses have been isolated susceptible animals are hard to find so that animal sera are not available

* R cketts æ are now considered by many to be viruses

Human serum from recovered cases gamma globulin or whole blood (from members of the same family) may be employed instead These sera are in general of little use in treatment but sometimes of great use in prevention or attenuation Note that viruses* are unaffected either by penicillin or the sulphonamides

Viruses although commonly described as ultra microscopic and filterable can be studied by means of the electron microscope and the elucidation of the relative mystery by which they are at present obscured should form the subject matter of the next great chapter in the conquest of infectious disease For all their minuteness they vary greatly in size thus the virus of psittacosis (275 mμ) is nearly thirty times as large as that of foot and mouth disease (10 mu) which itself is only about twice as large as the molecule of hæmoglobin The great difficulty in studying viruses is not all due to their small size however but also to the fact that they are complete parasites and can only be induced to proliferate on *living cells* of which the various components of the chick embryo are a favourite and readily accessible source much employed by investigators in recent years The technique is not difficult and we may hope for much from this method The disadvantages are that all viruses will *not grow* on the embryo and also that such passage on the bird may alter the nature of the virus just as passage through the cow sensibly modifies the agent of small pox They are resistant to 50 per cent glycerin which kills all ordinary bacteria unless *in spore form* The question arises are viruses really living organisms? because one type of virus has been prepared

* With very rare exceptions

in crystalline form without detriment to its activity The question must remain an open one It has been suggested that they are in fact a kind of enzyme which when introduced into a cell convert the contents into enzymes like themselves in other words that they are autocatalysts There is no doubt that they consist of protein which is an inseparable constituent of all living matter (and is found nowhere else) and that they are apparently capable of reproduction *They may constitute a bridge between inanimate and* animate matter For our purposes we may continue to speak of them loosely as organisms without being *led seriously astray*

But we must remember certain salient points about them As already pointed out they resist both penicillin and the sulphonamides It is perhaps also unsafe to rely on ordinary chemical disinfectants in the concentration normally employed to destroy them They certainly pass the hydrochloric acid barrier of the stomach as it would appear with ease and probably appear in the excreta far more frequently than is supposed though the latter are only regarded as infectious in poliomyelitis Virus also travels rapidly throughout the whole body and *in most* virus diseases we are probably dealing with a septicæmia or perhaps more accurately a viridæmia *This is surprisingly the case in such an ordinarily* benign condition as rubella where we must assume that the virus makes its way to the growing embryo to attack *characteristically* the developing tissues with dire results

Coccal diseases in general are characterized by short incubation periods sharp febrile reactions a

polynuclear response and a tendency to suppurative complications While infectivity is not so high as that of the viruses persistence of infectivity is notable and the carrier state is common Finally they are very vulnerable to drugs of the sulphonamide sulphanilamide group and to penicillin

Bacillary infections exhibit few fixed characteristics On the whole infectivity is low incubation periods tend to be long and the course of the disease to be protracted The carrier state is common While some like C diphtheriæ and H pertussis provoke little or no febrile response others like E typhosum illustrate perfectly the classical picture of a fever

The Nature of Bacterial Antigen

Though certain bacteria as we have seen have specialized as parasites on the higher forms of life disease need not follow infection Sometimes this parasitism is benign sometimes it may be actually beneficial as in the case of organisms normally resident in the intestinal tract That disease follows invasion by pathogenic bacteria at all is largely incidental almost one might say accidental for disease may result in the death of the ho t a major disaster from the invading parasite s point of view since it involves his own extinction We may well therefore ask what distinguishes the pathogenic organism from the harmless commensal and how do such pathogens set up disease?

Since we are still largely ignorant of bacterial biology the answer to this can only be tentative

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though it would seem as if in the last resort it might be a matter of chemistry Pathogens are distinguished from commensals by their invasiveness and their toxicity *Invasiveness* is the ability to spread widely through the body tissues *toxicity* is the capacity to produce local dysfunction or general bodily disturbance (The sum of both is described as *virulence*) While this distinction is reasonably valid we must remember that no organism is pathogenic at all times in every place and conversely that commensals out of their normal habitat may behave as pathogens A classic example is the B coli, which harmless in the lower bowel may set up serious or fatal disease if it should reach the peritoneal cavity bladder or kidneys Again organisms which are harmless in healthy surroundings may take on the character of pathogens should the tissues become devitalized by chill or injury We shall discuss the subject of bacterial variation later meantime when we refer to organisms unless otherwise stated we imply that they are pathogenic

As to how viruses set up disease it must be admitted that we are largely in the dark We cannot here invoke a local lesion discharging toxins into the blood That is not to say that local lesions do not occur but that we have no evidence of toxin formation The systemic symptoms then must it would seem be due to circulating virus How then does it cause a rise in temperature for example? Does the virus invade the cells constituting our (theoretical) heat regulating centre? If so one might imagine that in a certain number of cases that centre might be destroyed But this never seems to happen at any rate in cases

which recover though cases of chronic encephalitis lethargica exhibiting the Parkinsonian syndrome often show a very sluggish and restricted pyrexial reaction to infection Presumably it acts as a foreign protein In actual fact virus diseases do not depart appreciably from the pattern common to other infections as far as the clinical manifestations of disease are concerned apart from the fact that they are uniformly difficult to influence by treatment For most practical purposes therefore we may deal with them in the same way as diseases due to the larger organisms

Here we are on surer ground We know that certain of these e g the streptococcus staphylococcus C diphtheriæ Cl tetani and Cl botulinum produce soluble *exotoxins* so that if a culture be made in a fluid medium under appropriate conditions and filtered the filtrate will contain the exotoxin Many other organisms however do not in such circumstances produce such an exotoxin though this does not entitle us to say that they do not do so in the tissues If however they be killed the dead organisms will be found to be highly toxic so that an *endotoxin* is apparently released only after death In the tissues death of large numbers of bacteria must be a continuous process so that the contained endotoxins are consequently set free and absorbed Other bacterial products such as *lysins* which not only like the exotoxins destroy but in addition digest body cells and *aggressins* closely allied to endotoxins are also demonstrable but in so far as the clinician is concerned they are mainly of theoretical interest

Just as attack provokes defence so any foreign protein (and many non protein substances if linked

to protein) introduced parenterally into the human body will function as an **antigen** i e a substance which provokes the production of an **antibody** Antibodies are products which inactivate or neutralize the antigen The antigen may be a bacterial exotoxin in which case the antibody will be an antitoxin Or the antigen may be the red cells of another species of mammal in which case the antibody will be a lysin which lyses the invader cell Where the whole bacterium is used as an antigen the antibody which may then be described as antibacterial may consist of a number of different fractions corresponding to the different fractions of the bacterium thus there may be (a) anti endotoxins (b) lysins (c) precipitins —substances which when added to a solution of such bacterial products as are soluble will precipitate them and perhaps most important (d) agglutinins products which clump or agglutinate the bacilli and *especially if they are motile inactivate them* For convenience and clarity we will use the term antibody whether it refers to an anti exotoxin or to an agglutinin but there is one important point to remember anti exotoxins will unite with and neutralize exotoxins *in vitro* without more ado but certain antibacterial products require the presence of ***fresh complement*** if they are to produce their effects Hence the importance and often the dramatic effects of even small *transfusions in many infections And as these products are most specific* hence also the phenomenon of complement fixation

Advantage is taken of this antibody response to produce ready made antibody for use in human infections *A suitable animal usually (but not*

necessarily) the horse is inoculated at intervals with increasing doses of antigen (Following inoculation there is often a *negative phase* during which further antigen might prove fatal hence the carefully spaced intervals) In course of time the animal's blood becomes rich in antibody which it is found is contained in the serum The serum is therefore separated and sealed (under strict aseptic precautions) in ampoules where if kept in cold storage it should retain its properties for some time Dessication in vacuo is a more reliable method of preservation

The properties of a serum will of course depend on the antigen employed For instance where this is an exotoxin the resultant antitoxic serum may well be of paramount value in treatment On the other hand where the antigen is the whole bacillus anti endotoxic bodies may be present but they are *definitely less efficient in treatment than the former* They may be and are used in therapy but they are much more frequently employed in the identification of unknown organisms owing to the agglutinins and precipitins they also contain Since this reaction is specific agglutination in high degree will only occur with an organism identical with that used as the original antigen Where organisms of a given group which have more than one antigen share a common group antigen the phenomenon of co agglutination may occur in which case an absorption test may be necessary to distinguish them

What applies to the inoculated animal also applies to human beings This will be discussed more fully under Immunity Meantime we may note that the presence of antibodies in the serum is of great

value not only in diagnosis (for example the Widal reaction in enteric) but also in prophylaxis and treatment Any patient who has recovered from a given infectious disease will have specific antibody in his blood which reaches a maximum in convalescence and then declines Where the cause of the infection is unknown or impossible to isolate or not pathogenic to animals this antibody may be invaluable Especially is this so in those virus diseases e g measles where antibody persists throughout life Blood from recent convalescents or adults who have had the disease may be withdrawn the serum separated and stored in ampoules While not of great service in treatment the value of this human serum in prevention or attenuation cannot be exaggerated Care must be taken of course not to convey infection in this way consequently the Wassermann Reaction of such serum is tested as a routine before use Unfortunately in the acute stages of certain fevers (and in fact in measles) there is often a lipoid imbalance which gives a positive W R In such cases the patient s blood should be re tested after some weeks

Specific antibodies seem to be associated with certain of the globulins of the serum These globulins have been extracted to form what is called gamma globulin which volume for volume is much more powerful than the parent serum One danger of human serum is that it may convey the virus of infective hepatitis Perhaps gamma globulin may avert this risk

It is perhaps not generally realized that *whole blood* from say the mother of a child who has herself had measles during childhood can be just as efficacious

and can be withdrawn and injected *intramuscularly* with impunity provided there is no Rh incompatibility about which evidence should be sought In an emergency e g a child with an existing pneumonia exposed to measles this may save life Not only will an adequate quantity of such blood containing measles antibody if given early enough prevent or attenuate measles but the fresh complement reinvigorating an exhausted defence may turn the tide

BACTERIAL VARIATION

That bacteria can be made to vary greatly in virulence and to produce involution forms by suitable culture and subculture has long been known Thus by growing at temperatures unfavourably high or low by repeated subculture on unfavourable media and by other devices originally virulent organisms can be so attenuated as ultimately to lose all pathogenicity Conversely especially by passage through a susceptible animal virulence may be raised Not only can the virulence be made to vary but the very structure of the organism and its colonial form and appearance can be made to alter These three phenomena—alteration in (*a*) colony appearance (*b*) morphology of the organism and (*c*) antigenic structure have recently been intensively studied and are found to be intimately related

An old pure culture will often consist of two types of colonies one smooth and shining with clear cut rounded outlines the other rough and dull with jagged crenated margins With few exceptions the

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the disease in question (be it a frank an abortive or a subclinical attack) or to a carrier **Carriers** fall into two groups (*a*) the *contact carrier* who may be developing the disease or undergoing a subclinical attack or who may be immune in which case he may harbour the organism either *temporarily or indefinitely* and (*b*) the *convalescent carrier* a person who has recovered from a clinical attack of the disease but in whom the infection persists The carrier state does not develop in all infections not in whooping cough for example *nor seemingly in any* virus disease except possibly poliomyelitis and encephalitis lethargica As the carrier state also occurs in cerebro spinal fever we may say that the carrier is a common mechanism in the spread of acute infections of the central nervous system Of bacterial diseases scarlet fever diphtheria and enteric are outstanding examples of carrier borne diseases

It is obvious that organisms pent up in the body must reach the surface in some way or another before *spread* can occur This can only take place through the skin—via a scab or discharging abscess—or through one or other *of the natural orifices of the body* whether by way of the excreta or in the form of sputum or a discharge Thus meningococci in the cerebro spinal fluid of an actual case can be of no significance as a source of spread but in the nasal discharge of a carrier *may be an active* and dangerous source of infection It is therefore essential to know when and where organisms may be encountered and the paths by which they normally travel

Apart from the staphylococci etc commonly met with on the **skin** *specific infectious* agents are to be

"smooth" colonies consist of virulent organisms of normal appearance, forming good antigens for immunization whereas the "rough" organisms are relatively avirulent poor antigens for immunization and may show loss of capsules or flagella where these are features of their normal morphology

In the case of flagellated organisms such as those of the typho coli group two groups of antigens have been isolated those from the flagella the "H" and those from the body of the bacillus the "O" and the "Vi" The "H" antigens are of two kinds those specific to the organism and those common to other bacteria of the group they have nothing to do with invasiveness or toxicity which are the functions of the somatic "Vi" and "O"

The importance of these facts to our subject is two fold In the first place vaccines used as prophylactics should be made from fresh smooth cultures recently obtained from a case of the disease in an active phase Secondly in the diagnosis of such diseases as enteric the presence of "H" agglutinins merely indicates that an infection by or inoculation of one of a group of organisms has occurred in the past As will be seen later the "O" and "Vi" titres give us much more information

Sources of Infection

Apart from an important but limited group in which bacteria are introduced primarily from avian or mammalian sources to man all infection traces back to the human body whether to a sufferer from

a nasal discharge due to a streptococcus in one patient may give rise to impetigo in a second which may in turn cause scarlet fever in a third In addition it must be noted that we are still largely ignorant as to the extent to which vermin and wild or domestic animals and fowls may function as reservoirs or disseminators of infection

Few agents as we know are motile, and then only in a fluid medium They are passive and must be *transmitted* in one way or another The question therefore arises how is this achieved ?

Routes and Methods of Transmission of Infection

The old answer to this used to be flies food fingers and fomites and while hardly adequate this does include some of the most important mechanisms by which infection is spread especially if we enlarge the term flies to connote any insect To bring all possible mechanisms within the scope or a brief classification would be almost impossible It is easier to consider them in conjunction with the routes by which infection may enter the body since these are of necessity limited The following classification includes all the routes if not all the possible mechanisms and is sufficiently comprehensive

By the placenta — A child may be born with small pox Syphilis is frequently transmitted to the offspring

By auto-infection that is conveying organisms from one part of the body to another A classical

found there in smallpox and chickenpox as well as in impetigo furunculosis and so on Infection also reaches the surface in discharges from the eye ear nose urethra vagina or from a septic wound In a *large number of conditions infection is abundant in* the nasopharyngeal secretions whence it may be expelled in coughing and sneezing and it is expelled in the sputum in diseases of the lung In bowel or *kidney conditions infection may appear in the fæces* or urine whence it may pass into drinking water or via the fingers of a carrier into milk or other foods Even when an organism is apparently locked up safely *in the blood it may be released by the intervention of* a *vector* such as a louse flea or mosquito

Pathogenic agents may therefore contaminate the food or drink the feeding or sanitary utensils the *bedclothes pillows or nightgowns the books toys* or instruments with which the patient comes in contact (Such objects are known as *fomites*) Organisms may be found on the floor or in the dust and air about patients beds This aspect of the transmission of infection has received much attention in recent years since it has been shown that clean wounds exposed for dressing while the floor is being swept or an adjoining bed is being made may readily become infected by the organisms projected into the air by such activities It has therefore been recommended that floors blankets etc should be impregnated with oil and that no wounds should be exposed for a couple of hours after sweeping or bed making has been carried out In connection with this sort of cross infection it is important to remember that like may not necessarily breed like in the sense that

function actively in the spread of summer diarrhœa enteric and other intestinal infections by alighting on infected stools and subsequently on food

By inhalation —Every time we speak and particularly when we cough or sneeze we project minute invisible *droplets* of tracheal nasopharyngeal or buccal secretion into the air often as far as ten or perhaps twenty feet Where infection is heavy in the nasopharynx as it is in so many infectious diseases the result will be obvious Droplets may impinge on the lips or nares of another patient and may also be inhaled perhaps even more dangerous they may be directed into milk or other foods where the organisms may multiply freely Quite apart from droplets dust may contain organisms not only the tubercle bacillus but streptococci and others and in some cases it seems that they are quite likely to be inhaled in sufficient quantity to produce disease Finally it must be remembered that some authorities consider smallpox and perhaps chickenpox to be transmissible through the air even over quite considerable distances

By ingestion —Contaminated water or food may be swallowed as in food poisoning enteric and so on Apart from such obvious intestinal infections however it is important to remember that diphtheria streptococcal sore throat and other conditions can be conveyed in this manner especially by milk

By inoculation —The intact skin is an impassable barrier to all micro organisms but quite a small abrasion of the epithelium may destroy this integrity The fæces of a louse carrying the Rickettsia of typhus may be inoculated by scratching in fact this is probably the normal method of infection Vaccination

example of this is the transfer of gonococci from the urethra to the eye but this is in practice a rare occurrence Far more common though seldom stressed is the transfer of pathogens from the nose to other parts of the body The nose frequently harbours both staphylo and strepto cocci and may harbour meningococci and C diphtheriæ as well as—in season so to speak—such viruses as those of influenza the *common cold poliomyelitis and so on People finger* or rub their noses unconsciously following which they may scratch the skin and cause a boil or rub their eyes and set up a blepharitis or *conjunctivitis* or develop a septic finger Diphtheria of a wound has undoubtedly been caused in this way Even food especially liquids like milk or soup may be similarly contaminated In this connection the borrowing of *another person s handkerchief is a most dangerous* and unhygienic practice

By direct contact —As for example in kissing a frequent channel as between adult and child and not uncommon as between adult and adult The *venereal diseases are typical examples of infection by* direct contact

By indirect contact —A most important mechanism of spread Just as the insect pollinates flowers so the hands of the attendants are the supreme agents in bridging the gap between the source and the recipient of infection Fomites such as a toy spoon a borrowed handkerchief or a sucked pencil turned over to a colleague at school may also play a decisive part The clothes of a person suffering from smallpox have more than once *infected* and *killed* workers who handled them in a laundry Vectors such as flies may

SECTION II

RESULTS OF INFECTION—DISEASE

WHEN organisms invade the body much as when an army invades a country, the result will depend on three variable factors (*a*) the **mass** or number of the invaders (*b*) their **virulence** and (*c*) the body **resistance** (which by the way is by no means synonymous with athletic fitness) If (*a*) multiplied by (*b*) is less than or equal to (*c*) no appreciable result may follow though immunologically and epidemiologically the outcome may be very important since the effect will be to still further heighten resistance The more nearly (*a*)×(*b*) approximates to (*c*) the more likely is what is called a subclinical attack to ensue—perhaps a transient afebrile catarrh which while happily increasing resistance as we have noted may also act as a serious and unrecognized agent of spread On the other hand if (*a*)×(*b*) is greater than (*c*) whether (*c*) be normally low or depressed momentarily by cold starvation or fatigue an attack of disease will follow in due course

Not be it noted immediately Between the moment of infection and the first sign of disease there is invariably an interval known as the **incubation period** This varies with the different infections but is within limits remarkably constant for any given infection What happens during the incubation period is still a matter for speculation Many believe that the

is achieved by a similar scratch Perhaps the most rapidly fatal of all, streptococcal infections sustained at post mortems arise in this way The puncture that pierces the skin but does not cause bleeding is the most dangerous of all

In practice many factors influence the spread of infection For example in a cold moist climate such as ours droplet spread in winter is much more frequent and serious than in summer, since out of doors it is almost negligible Thus one can go for a walk with a friend suffering from a cold with very little risk of catching it whereas to spend the evening around the fire with him or travel with him in a stuffy railway carriage is to make almost certain of getting it

cases results in the condition known as the *febrile state*

The febrile state is an omnibus term to describe the customary disturbance of the normal functioning of the heat regulating centre the central nervous circulatory digestive and excretory systems which accompanies *most* but not necessarily *all* acute bacterial invasions The more intense the toxæmia

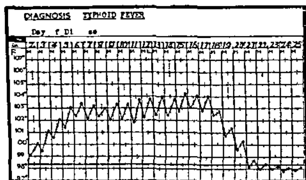


Fig 1—Continuous pyrexia Termination by lysis

the sharper as a rule the febrile reaction This must now be considered in its component parts

Heat regulating mechanism—pyrexia—Our bodies are warmer than our surroundings Heat is generated by burning foodstuffs and lost via the skin and breath Body temperature is fairly constant and varies from 97 F to 98 4 F in health and at rest (The rectal temperature is about 1 F above that of the mouth axilla or groin) At the onset of fever *corresponding to the entry of the organism or its toxin into the blood*

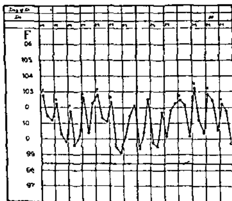
interval is occupied in developing an allergy to the infective agent in question but we may assume without hurt that the organisms are endeavouring to reach a favourable site on which to proliferate while the body is attempting to dislodge them Each infective agent for reasons which we do not understand has its own incubation period to which within certain limits, it faithfully adheres The more virulent the organism and the lower body resistance the shorter the incubation period *for that particular disease* and the sharper the attack the more feeble the organism and the higher the resistance the longer the incubation period and the milder the attack Thus a child exposed to measles will normally sicken in about ten days and produce a rash in about a fortnight whereas if he has been partially protected by serum he may not develop a rash for three weeks and the attack may be very mild

But that is not to say that diseases with short incubation periods are necessarily more serious than those with long there is in fact no such relationship Thus a cold may have an incubation period of two to three days while for smallpox it is fairly consistently twelve *

Should the organisms prevail in course of time they arrive at a favourable site—the *site of election* There is a sudden efflorescence as they set up house and proceed to multiply The familiar phenomena of inflammation appear in the part attacked—*the local lesion*, as it is called to distinguish it from the generalized effects of the resultant toxæmia which *in most*

* Note well that during the incubation period the patient is *not infectious*

sponging is permissible It may be and often is the first indication of an impending illness or of a com plication in an existing disease The temperature should therefore be carefully recorded twice daily on a chart No patient with pyrexia should be allowed to get up While the varieties of pyrexia are perhaps mainly of academic interest they are useful in de



F g 3— Inverted pyrex a (pulmonary tubercolos)

scription and may often afford valuable diagnostic clues Thus inverted pyrexia would suggest either late tuberculosis or some suppurative lesion in which drainage is adequate during the day but not at night Persistent pyrexia even though unaccompanied by any other sign (P U O) is a warning that an active infection such as enteric tuberculosis enclosed suppuration etc is in progress and the student must not desist until he has discovered the cau e Certain temperature charts are so characteristic that

the patient feels chilly and shivers—often violently—rigor The muscles of the skin contract producing "gooseflesh" and narrowing of the arterioles so that heat is conserved Sweating—a potent source of heat loss also ceases the skin becomes hot and dry and the temperature mounts A rise in temperature is called **Pyrexia,** and the following varieties are distinguished

(1) **Continued** *—where the temperature goes up

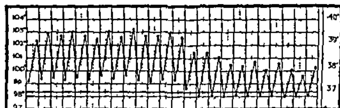


Fig — Remittent pyrexia becoming later intermittent

and remains up with a morning and evening variation of less than 2° F

(2) **Remittent**—as (1) but the morning and evening variation is greater than 2° F though never reaching normal

(3) **Intermittent**—where the morning reading is *normal* or *below* but the evening reading is *above*

(4) **Inverted**—where the morning reading is higher than the evening

Significance of pyrexia —With the rarest exceptions pyrexia at rest implies toxæmia and therefore microbic activity It is probably a defensive reaction and should never be reduced by drugs though if very high

* Continued fever is often used as a synonym for enteric

muttering to himself He sees and hears things that do not exist (hallucinations of sight and sound) and he may develop unfounded beliefs (delusions) such as that the nurse is an enemy with the result that he may attack her (*Delirium tremens* is the classic example where alcohol is the toxin) He nearly always tries to get out of bed and as a result of the compulsion to escape may readily jump out the window Hence the importance in any condition where delirium is to be feared of nursing the patient on the ground floor under constant supervision

Stupor may precede succeed or alternate with delirium Here the patient lies supine He can be roused and will swallow reflexly but relapses as soon as the stimulus is withdrawn He is incontinent *Coma* is a stage further than stupor the patient is quite unconscious feels no pain and cannot be roused He is of course incontinent *Coma vigil* is a variety of coma commonly seen in the *typhoid state* (see p 168) Finally note that in small children almost any febrile condition may start with a convulsion

Circulatory System

A rapidly beating heart and a frequent pulse of good tension are typical of the febrile state In most cases the pulse rate and temperature rise concurrently but in certain conditions disproportion is manifest e g in enteric where the temperature is high but the pulse rate is slow Whenever an increase in intracranial pressure occurs the pulse rate is also

an examiner may expect the student to recognize them at sight (see p 345) As a rule, only in children does excitement induce pyrexia the mere fact of removal to hospital in an ambulance is enough to send the temperature up a degree *Malingerers* and hysterical patients often simulate pyrexia by friction with the tongue, or the prior application of a hot water bottle to the axilla Where this is suspected it is advisable to record the temperature simultaneously in the mouth and both axillæ

Central Nervous System

Headache and mental dullness are invariable in fever of any degree In severe toxæmia movements

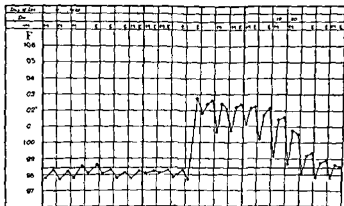


Fig 4 —Onset of complication

and speech may be tremulous and delirium ensue In *delirium* the patient is noisy mentally confused and

Genito Urinary System

Dehydration is a prime characteristic of fever the body is holding on to water and thus the urine is diminished concentrated and highly coloured Albuminuria is common and need cause no anxiety On standing there is a thick red brown deposit of urates which should not be confused with the smoky urine of moderate hæmaturia In severely toxic states where the circulation is failing suppression often supervenes although the kidneys may still be efficient

Course of Fevers

Fevers are (arbitrarily) divided into the following stages which are often ill defined and in any given disease often absent —

Incubation period —From the moment of infection to the appearance of the first sign or symptom of the illness

Stage of invasion —From the first sign of illness to the first characteristic stigma of the disease (Where this is a rash it is often called the prodromal period It is also the stage of wrong diagnosis as transient *prodromal rashes* often appear)

Acme Fastigium, or Stage of Advance is that period during which the fever is most pronounced and all signs and symptoms are at their worst It may often correspond to the duration of a rash

Stage of decline —From the first sign of abatement of the fever to its disappearance The temperature

slowed—for example in meningitis and here also disproportion will be evident In other conditions where pyrexia is not a notable reaction to the toxæmia, such as diphtheria disproportion may be extreme inasmuch as the pulse may be uncountable while the temperature is subnormal Provided the many factors (such as emotion) which cause tachycardia can be eliminated the pulse rate is probably a more sensitive index of toxæmia than the temperature This is exemplified in early tuberculosis The pulse rate should be noted at least as carefully as the temperature

In severely toxic states such as hypertoxic diphtheria the tension of the pulse may steadily fall and the rate increase to double or more Next the extremities go cold and the patient lies semi conscious as the peripheral circulation slowly fails This throws still more work on the heart so that unless the toxæmia is relieved it fails in its turn

Digestive System

Anorexia nausea and vomiting are common occurrences in febrile patients The patient is dehydrated Thus the salivary secretions are diminished the breath is foul and the mouth dry In prolonged or severe fever the tongue becomes dry brown and wrinkled a dry scum—*sordes*—forms on the lips a film coats the palate and particles of food epithelial debris and dried up secretions line the gums and collect between the teeth Constipation is usual diarrhoea rare

fever a transient bacteriæmic stage occurs before the organisms settle down in the tissues for which they have a predilection The blood it would appear is normally bactericidal in high degree unless immobilized by thrombosis when it may serve by contrast as a culture medium This occurs most strikingly in puerperal fever where the thrombosed veins of the broad ligament may act as limitless reservoirs of organisms which pour unceasingly into the blood stream until the patient succumbs but it also occurs in some degree around any local lesion, which should consequently be handled gently lest the natural barriers be broken down and the infected thrombi be broadcast generally through the system The terminology employed to describe organisms in the blood is somewhat confusing Where the condition is very transient the word bacteriæmia is employed where it is relatively more permanent the term septicæmia is introduced

Septicæmia as normally understood implies that an organism of high virulence is circulating and possibly multiplying in the blood stream The most common organisms are the streptococci staphylococci pneumococci B coli and the B Pfeiffer Major lesions of high mortality are likely to ensue—almost always pneumonia perhaps general peritonitis empyema purulent pericarditis or meningitis or indeed all the foregoing at once So called **pyæmia** is a condition in which metastatic abscesses occur

In many acute fevers however there is further a fulminant or hæmorrhagic type described in which the disease begins perhaps with violent rigors alarming pyrexia and tachycardia followed rapidly by a

may fall by *crisis*—within twelve hours, or by *lysis*—over a period of days

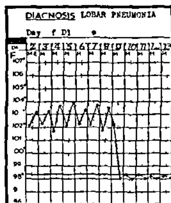


Fig 5—Termination by crisis

Stages of convalescence —From cessation of symptoms until full normal health is regained

Bacteriæmia and Septicæmia

Instead of remaining localized in the local lesion and pervading the blood stream with their toxins the organisms may themselves pass into the blood This is comparable to an invading army securing control of the railway system of the country attacked In most if not all virus infections such as measles smallpox chickenpox etc the virus is present in the blood during the early stages In enteric certainly and usually also in pneumonia and cerebro spinal

general immunity This as noted has nothing to do with athletic fitness it is related purely and simply (as far as we know) to previous experience of the invading organism which is why the young and vigorous may often succumb to an infection which the aged and enfeebled repel

Immunity may be *natural* or *acquired* **Natural** immunity is exemplified first of all in *species* immunity thus the goat is immune to tuberculosis whereas the cow is very susceptible Presumably the biochemical environment in the goat is distasteful if not indeed inimical to the mycobacterium while in the cow it is very agreeable Again most animals are immune to syphilis which attacks only the higher apes and man *Racial* immunity is less clear cut nevertheless the incidence of tuberculosis amongst the Jewish race is very low whereas amongst the Celtic race it is very high Natural immunity is inherited though sometimes only of short duration thus babies under six months rarely contract measles since (provided their mothers have had the disease) their inherited antibodies protect them As antibodies are also excreted in the milk in breast fed babies immunity is higher and lasts longer—one of the many cogent reasons why breast feeding should be adopted **Acquired immunity** may be *active* or *passive* Active immunity may first of all be acquired by an attack of the disease in question—the normal and time honoured way But as it is usually painful and often hazardous it is not surprising that methods have been adopted to secure the same result with a minimum of discomfort and risk

Nature herself has provided a clue It is well established that children are highly susceptible to

hæmorrhagic rash, at which stage death curtails the process This would seem to indicate either that the organism in question is of exceptional virulence or alternatively that an organism which normally does not do so has broken into the blood stream

It is often rather difficult to know how diagnosis is made in such cases unless by inference since one petechial or ecchymotic rash is very like another and associated symptoms may not be prominent When ever such hæmorrhagic rashes appear in the infections the outlook must be regarded as extremely grave with the rarest exceptions In such fulminant cases hæmorrhages in the cortices of the suprarenals are also of frequent occurrence

Results of Infection—Immunity

Immunity is the ability of the body to resist invasion by any given organism Recovery is due as we have seen to the appearance of the appropriate antibodies in sufficient quantity in the blood When the invasion has been repelled these antibodies persist in the blood in some cases only for a short period but in other cases for many years if not for life Why this should be we do not know such (mostly virus) diseases as confer a lifelong immunity are always associated with a lymphocytosis though all diseases characterized by a lymphocytosis do not confer lasting immunity It is possible that in some diseases virus persists in the cells for life thus constituting a perma nent source of antigen In any event the measure of the antibody content of the blood is the measure of

(among other antibodies) being thus produced The antigenic properties of many organisms are not abolished by death they are therefore first killed and then suspended in saline to constitute a *vaccine* In such diseases as enteric plague etc these vaccines may produce satisfactory immunity for some years

Where filterable viruses are concerned the problem is more difficult since they cannot be cultivated except on living tissue and it has hitherto proved extremely difficult to separate them from that tissue The virus therefore has had to be presented whole together with the tissue with which it is associated This may have been the contents of a pock as in the case of smallpox transmitted to a calf (vaccination) or as in Pasteur's classic antirabies inoculation the dried spinal cord of the rabbit in an emulsified form The modern method of chick-embryo cultivation however coupled with the use of the high speed centrifuge makes it possible to obtain the virus in pure form The next step is to attenuate it without killing it (since dead virus appears in most cases to be inert) and to work out a satisfactory system of dosage This then is the dilemma of the immunologist the virus must be alive but attenuation and mensuration of dose are uncertain if attenuation be insufficient the result may be disastrous if it be excessive no immunity may be produced

Under the spur of military necessity much work on this problem has been carried out during recent years so that nowadays a number of different virus vaccines are in fairly common use A yellow fever vaccine was extensively employed in the American Army during the war and vaccines of the A and B strains

diphtheria whereas adults even those who have never suffered from it have appreciable quantities of diphtheria antitoxin in their blood This is brought about by the *mechanism of the submorbid dose* and the subclinical attack A dose of infection not sufficient to set up disease is sustained and repelled *A small quantity of antitoxin is required as a result* Later on a further and it may be larger dose of infection is experienced with a corresponding gain in *the antibody content of the blood Later perhaps a* massive and virulent dose may be survived with no more than a minimal bodily reaction so slight as to pass unnoticed *These are the* **subclinical attacks** unrecognized but potent factors in immunity production They must occur in most if not all infections seeing that antibody commonly appears in the blood in cases where there was none before and since only antigen can produce antibody antigen must have been somehow administered As no *clinical* attack has been observed the inference is obvious

Devices to secure antibody formation are concerned with procuring the highest possible immunity with the least possible discomfort to the patient within a reasonable time Where a soluble exotoxin is available this is relatively simple The toxin itself can be employed but it is more usual to try to curb its virulence by prior treatment if possible without diminishing its antigenic powers Such toxins are then described as toxoids Formalin is a very useful agent in curbing toxin and is widely used Heating or prolonged storage seem to have a similar effect Where no exotoxin is available the whole bacterium may be employed an anti endotoxin

a friendly power masks be supplied in huge quantity (passive immunity) so quickly as to make the erection of their own factories superfluous then as soon as the masks have worn out or perished the country will be vulnerable (susceptible) again It is important to remember therefore that in serum treated diseases the sooner the serum is administered the better the immediate outlook but the more susceptible the patient will subsequently remain since his immunizing factories may hardly have the foundations laid Much the same will apply in the case of diseases treated by the sulphonamides or penicillin If the condition is aborted very early in its career then obviously little immunity can be expected to develop whereas if these drugs are withheld until a late stage a fair if not a reasonably high measure of immunity should result

To summarize **Immunity** may be natural or acquired **Natural** immunity may be inborn in the species or race when it is lifelong or inherited from the parent when it may last from six to twelve months or more **Acquired** immunity may be *active* or *passive* Active immunity results from the impact of the antigen on the body defences following either a clinical attack of the disease or repeated subclinical attacks or alternatively the artificial introduction of live or dead bacteria or their antigenic products **Passive** immunity results from the acquisition of ready made antibody from an animal or person in the form of a serum—human or animal—or of a placental extract Since antibodies are associated with certain of the globulin fractions of serum protein 'immune globulin as it is called is a further refinement in the preparation of antibody from human serum

of influenza are now under extensive trial here and in the U S A No doubt in time vaccines of most of the viruses that flourish in the *upper respiratory tract* will be readily available and provided that they confer a sufficiently durable immunity to make their employment worth while should materially reduce the number of such infections from which at present we suffer so *frequently*

Passive immunity is conferred by a dose of ready made antibody contained either in an immune serum, whether animal or human or—in essence the same thing—in a placental extract But since such anti bodies are excreted by the recipient in about three weeks *passive immunity is essentially transient*

The whole position in regard to immunity may be aptly compared to that of a country attacked with gas bombs from the air Should such attacks have been occurring sporadically and at intervals on a minor scale (subclinical) over a period of years, most of the population will have wisely provided them selves with masks (antibodies) and factories for their production will be in existence easily expanded if necessary The country as a whole will therefore show a high degree of active immunity On the other hand if a mass attack comes as a surprise on an unprotected populace serious dislocation will ensue (attack of disease) until masks (antibodies) and factories to make them appear in sufficient quantity otherwise the inhabitants will be exterminated Should the country weather the onslaught such masks and factories will remain in being for so long that the country will be for all practical purposes actively immune On the other hand if through the offices of

and last methods are falling into disuse The intravenous is the route employed when speed of action is essential in the absence of an available vein the intraperitoneal route may be used as absorption is almost as rapid The intramuscular is however the common route employed absorption is reasonably rapid being fairly well established in about six hours The muscles in front of the thigh are best if serum be given between the scapulæ or into the buttocks the patient cannot lie comfortably in bed Subcutaneously serum is much more painful and absorption takes several days In general the serum should be warmed to blood heat before administration whichever route is chosen but should not be placed in boiling water else it will be coagulated

Serum Sickness and Anaphylaxis

Animal sera naturally contain proteins foreign to the human economy When such sera are employed therapeutically because of their antibody content the antibody will have an immediately beneficial effect on the patient s clinical condition None the less the associated foreign proteins will act as an antigen themselves an antigen which behaves in the reverse of the normal way Depending on the age and idiosyncrasy of the patient certain reactions may follow Of these much the commonest is **serum sickness** This usually appears in from seven to fourteen days When fully developed it may consist of (*a*) high pyrexia (*b*) albuminuria (*c*) an inflamed throat with cervical adenitis (*angina redux*) (*d*)

Tissue immunity—It is well known that the various organisms have a predilection for certain tissues e g the B typhi for the Peyer s patches of the small intestine and the B anthracis for the skin Some believe that if these tissues be rendered immune the whole organism will be immune They therefore administer typhoid vaccines orally and render guinea pigs immune to anthrax by rubbing attenuated bacilli into the skin That local resistance is the sole or dominant factor in the subsequent immunity is not clear since antibody is demonstrable in the blood This is only to be expected since the connection of the skin with the antibody manufacturing tissues (believed to be the reticulo endothelial system) is very close

On the other hand certain tissues are, for all practical purposes, immune to certain infections Thus though diphtheria bacilli may invade the whole upper respiratory tract invasion of the œsophagus almost never occurs Again though the tubercle bacillus may attack practically any tissue it leaves muscle severely alone Babies under six months repel the streptococcal scarlet fever but are notoriously susceptible to the streptococcal erysipelas These phenomena together with the age groups favoured by different infections are but a few of many problems still awaiting solution

Administration of Serum

Serum may, in theory be given in five ways—subcutaneously intramuscularly intravenously intraperitoneally or intrathecally In practice the first



PLATE I

[illegible]

[illegible]

arthritis (*e*) puffy œdematous swellings usually of the face and (*f*) serum rash all of which may persist for a week The serum rash may be first scarlatiniform or morbilliform, it may then pass through an urticarial stage and finally end up as an erythema circinatum' Such extensive reactions are rare and usually occur in adults who are of the 'allergic or asthmatic diathesis hypersensitive to horse serum Albuminuria slight pyrexia and an urticarial rash are the common signs, the rash usually dominating the picture (see Plate I) It may appear and disappear with great rapidity over a period of days In adults especially it may itch unbearably small children usually ignore it However generalized it may be there are almost always islands of normal skin to be seen It is noteworthy that severely toxic cases of diphtheria rarely react violently to serum (probably because of circulatory depression) and that when they do the prognosis is usually good

Anaphylaxis and hypersensitiveness—acquired— Once a patient has been injected with an animal serum whether he subsequently develops serum sickness or not he is liable to react excessively to any further injection of serum from the same species of animal This sensitized state develops about ten days after injection and persists though gradually diminishing for life Should a second dose be given say three days after the first nothing will happen Or if many years have elapsed perhaps only an *accelerated* reaction—i e serum sickness appearing within two or four days—may follow But if the second dose be given perhaps a fortnight after the first alarming results may follow at once or within

an hour (*immediate* reaction) The symptoms are usually restlessness dyspnœa rigors vomiting and collapse, and appear to be due to spasm of the un striped muscle throughout the body Fatalities have been recorded

In laboratory animals fatal anaphylaxis can readily be induced and immediate death can be produced by intravenous injection though each species of animal dies in a different way From such experiments it has been established (*a*) that only a minute dose is required to sensitize the animal (*b*) that the shock producing dose must be vastly greater (perhaps 1000 times) than the former (*c*) that the anaphylactic state develops gradually and not suddenly (*d*) that it can be transferred from a sensitized to a normal animal by injecting the latter with serum from the former and finally (*e*) that the reaction can be abolished by anæsthesia or modified by adrenalin This is a summary of the known facts to summarize at reasonable length the theories accounting for them would be impossible

Congenital anaphylaxis is an exceedingly rare but dangerous form in which death may follow even the smallest dose of serum *however administered* It is sometimes very definitely associated with asthma hay fever or other manifestations of the allergic state consequently such patients should be approached with the greatest caution

Thermal reaction —Intravenous serum is often followed by a rigor which however alarming is rarely fatal Since such rigors may follow the intra venous injection of many protein and even non protein substances it is not specific to the serum as

and far less likely to produce severe reactions than the unconcentrated serum of former days was prone to do and should always be used when serum is to be given intravenously The process of concentration is undergoing continuous refinement and improvement Where a patient is known to be dangerously horse sensitive serum from another animal e g the goat may be substituted Some of the larger manufacturers carry a stock

Serum rash and sickness* are (temporarily) relieved by an injection of $\frac{1}{4}$ or $\frac{1}{2}$ c c of a fresh solution of adrenalin chloride $\frac{1}{1000}$ severe reactions can be prevented or aborted by the same means Menthol or calamine ointment or cream to the rash are very soothing Before the administration of any but human serum enquiry should always be made for a *history of asthma or of previous animal serum* (Adrenalin should always be to hand) Should the history arouse or confirm suspicion a drop of the serum may be placed in the conjunctival sac or 0 2 c c of a $\frac{1}{10}$ dilution of concentrated serum in saline may be injected intradermally Should no reaction ensue within half an hour the remainder of the serum should be given intramuscularly Should a reaction (other than an alarming one) occur the serum can be administered in half hourly doses beginning with 0 5 c c and doubling up and standing by with adrenalin alternatively following the first 0 5 c c one may wait four hours and give the whole dose intramuscularly with adrenalin The former is perhaps safer the latter is more humane

* Benadryl and similar products have been reported as very effective in prevention and treatment

such, at all It is sometimes due to thermophilic organisms or to the nitritoid reaction

Tissue sensitivity —In sensitized animals or patients apart from other manifestations gross œdema and swelling may occur around the site of an injection Necrosis and sloughing may follow This is known as the *Arthus phenomenon* It is extremely rare

Summary —Individual idiosyncrasy plays a large part in serum reactions Some people do not react at all to the first injection but most do so on re injection Mild first reactions are the rule and though asthmatics are often sharp reactors often they are not In many hundreds of thousands of injections of which I have knowledge three ¹ serum deaths have occurred a fourth was doubtful All followed intramuscular or subcutaneous injections In no case was there a history of previous serum and *only one was a recognized asthmatic* All three showed the same picture—a normal interval of about ten minutes followed by sudden extreme and fatal dyspnœa—probably due to spasm of the bronchioles or of the pulmonary arteries Two cases had had only 0 5 c c subcutaneously Admitting the difficulty of completely excluding previous sensitization one is tempted to say that those who have once had serum although they may exhibit very severe and alarming reactions will never die of it The contrary is how ever the accepted teaching

Treatment and prevention —Since the antitoxic fraction is linked to the pseudoglobulins of horse serum the albumen and euglobulin are nowadays precipitated or digested out The resultant con centrated or digested serum is smaller in bulk

SECTION III

SPREAD AND CONTROL OF INFECTION

Elementary Epidemiology

EPIDEMIOLOGY is the study of the incidence and spread of infectious disease An epidemic is a great increase in the incidence of any given infectious disease occurring in any given place during a limited period of time Where a disease normally occurs in any particular locality it is said to be **endemic** When an epidemic is world wide it is described as a **pandemic** Epidemics are also described as **seasonal** when they recur regularly during a certain season or **solitary** when they arise irregularly irrespective of the time of year There is by the way no recognized dividing line between a *seasonal prevalence* and an epidemic—it is a matter of opinion and degree

If a graph of an epidemic be made plotting the number of cases along the vertical and the time in weeks or months along the horizontal it will be found that the result is cone shaped like a mountain peak The summit may be a point or a brief plateau That is there is a rise to a maximum and then a decline Occasionally there may be foothills on either side of the peak showing that preliminary and subsequent flare ups have occurred but they are usually dwarfed by the peak proper The steepness will vary with the various diseases depending on the ease and rapidity with which the condition is transmitted

In practice these procedures may not always be possible There is of course the alternative in mild cases (other than diphtheria) of giving no serum at all Conversely in really severe cases, especially of diphtheria serum must be given as soon as possible The risk of death, if it is withheld for any appreciable period is immediate, the risk of death from serum is very remote Better give adrenalin, and the serum intramuscularly at once (Never use the intravenous route if in doubt) In any event skin reactions are unreliable they are often pronounced when an intramuscular injection is uneventful and merely accentuate the dilemma of the physician Some authorities believe that, in those rare congenitally hypersensitive cases (which are mostly to be feared) even $\frac{1}{50}$ c c of serum may actuate the ' trigger ' mechanism in question and that it is quite impossible to decide on a safe commencing dose The moral is when in doubt to have adrenalin immediately to hand to give some *initially* and to keep the patient under close observation for half an hour since he may appear normal for 10 or 15 minutes At the least suggestion of dyspnœa or flush or pallor give further adrenalin at once and treat for shock

carrier rate (*b*) the epidemic comes to an end before all the mice have been attacked (*c*) during the outbreak there is a rise in the immunity of all mice including those who may subsequently contract the disease and (*d*) if when the epidemic is dying out fresh mice be introduced to the community a flare up will occur

There is however a vast difference between such an experiment under artificial conditions with one particular organism and what occurs in a human epidemic under natural conditions Normally in human infections during non epidemic periods there is an interplay of certain *factors* which results in an equilibrium expressed as say an average of a given number of cases of any given infection per year Disturbance of this equilibrium as a result of a material alteration in any of these factors may result in a swing of the figures in either direction The factor in question are (*a*) the infective agent (*b*) the manner in which infection is conveyed (*c*) the immunological state of the community (*d*) their environment and (*e*) the weather

To consider first the **infective agent** we may say in general that the smaller it is the more infectious it proves Thus virus diseases are of all the most infectious Even as between viruses however there are wide variations in infectivity—contrast smallpox with mumps for example Furthermore even any given virus may display varying degrees of infectivity and virulence at different times (These characters are not synonymous though often assumed to be) Thus there may be an outbreak of influenza—extremely infectious but mild Or it may be less than

thus for measles it might resemble a skyscraper while for whooping cough it may be a gentle slope (see below)

Why do epidemics occur? Since the earliest times physicians have speculated on this question

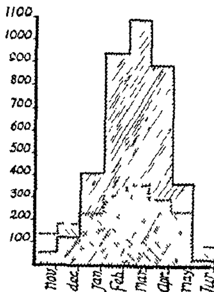


Fig [illegible] — Graph of simultaneous epidemics —Measles (continuous line) and Whooping Cough (broken line)

and it has not yet been completely answered Much experimental work has been done especially with mice using the organism of mouse typhoid From these experiments *some interesting facts have been* elicited —(*a*) before an epidemic there is a rise in the

sharply week by week by double or more until the acme is reached and decline again occurs And even decline may set in long before the number of susceptibles has been completely exhausted It is certainly quite possible that biological alterations in the infectivity of the agent may account for the rise and fall of many epidemics though it seems unlikely to be the sole factor involved

The question is often asked What becomes of the infectious agents of diseases like measles or influenza which die out apparently completely between epidemics? Where do they hibernate so to speak and where do they come from to start new epidemics? The answer is that we don t really know One theory is that cases—often atypical or subclinical—continue to occur between epidemics but usually escape recognition Another is that the agent is permanently resident in some species of bird or mammal among which it periodically sets up epidemics that are later communicated to man In the case of influenza for instance it has been shown that the virus of swine influenza inhabits the lung worm which is a parasite of the pig Infected pigs void and other pigs ingest the parasite When autumn chills depress the animals resistance an outbreak of swine influenza occurs to be followed in due course by a human epidemic This is a plausible theory which may well be correct

Next we must consider the vehicle or manner in which the disease in question is normally spread However infective the organism it can only exploit its opportunities it cannot create them In a scattered community of hermits vowed to perpetual solitude and silence diseases communicated by

usually communicable though severe when contracted. We may even distinguish between the constituents of virulence that is between *toxicity*—where lesions may be circumscribed but the constitutional disturbances profound—and *invasiveness* where lesions may be widespread but constitutional disturbance slight. We know that the 'virulence' of organisms may be exalted by passage through a susceptible animal or by growing on certain media at favourable temperatures and that it may be depressed by the contrary procedures but we cannot say this of their infectivity. Indeed of their biology in the natural state we are largely ignorant though an analogy from another field may be of interest. The lemming is a small Scandinavian rodent reasonably common in normal times. But once in every five years or so when (presumably) conditions for propagation are extraordinarily favourable there occurs what can only be described as a plague of lemmings. In their thousands perhaps in their millions they stream down the mountain sides blindly seeking the sea into which they plunge without hesitation to swim frantically away from land until they drown. While this may be an extreme instance of *élan vital* that similar and equally senseless efflorescences of bacterial life may occur cannot confidently be denied.

That something of the sort occurs in measles seems highly probable. Following an epidemic for roughly eighteen months the disease is relatively uncommon. As the number of susceptibles is rapidly increasing it might be expected that more and more cases would gradually appear. But this is not the case. When it comes the rise is sudden the incidence increasing

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droplet spray, would be most unlikely to spread Even a pandemic of influenza would probably pass them by But as their sanitation would be primitive an epidemic of enteric via their drinking water might easily arise This is the reverse of the situation in cities where thanks to modern sanitation millions may live in close proximity with a very small risk of enteric but where owing to our aggregations in schools trams theatres shops offices etc the importation of even a single case of virulent influenza at an appropriate time might result in hundreds of deaths

Where the chain of causation is long and involved the absence of one or other vital link will naturally prevent spread therefore such diseases are much less likely to become epidemic Thus in the case of plague in addition to the B pestis the black rat and the *cheopis* flea are essential Further the organism must inhabit the blood of the rat which itself must live in such close contact with humanity that the flea can readily convey the pestilence to man In the old grossly overcrowded wooden houses of which the London of Charles II was largely composed rat infestation was understandably very heavy hence one reason for the Great Plague Modern housing alone is a formidable deterrent to a repetition of this disaster

Again apart from the infectivity of the organism the clinical character of the disease must influence the ease and rapidity with which it spreads In early measles and mumps for example the causative agent abounds in the mouth and nasopharynx but whereas in mumps comparatively close contact with the

patient s secretions or fomites is necessary for trans mission in measles the characteristic violently ex plosive coughing and sneezing broadcast infection far and wide

Infection may be broadcast but will it take root? This brings us to our second major factor—the **susceptibility or immunity** of those exposed considered as individuals or as a herd In virgin soil infection will spread like wildfire Witness the ravages of tuberculosis among the negro slaves first brought into contact with it in North America or the celebrated epidemic of measles in the Faroe Islands (where measles had been unknown for a generation) in which not a grandparent nor a grandchild of the susceptibles escaped It is a readily observable fact that country children brought to town or sent to school proceed to contract the common exanthemata one after the other Again where cerebro spinal fever occurs in barracks it is a safe prophecy that the victims are country lads— unsalted as the phrase goes—who have encountered the meningococcus for the first time The town dweller has had his frequent submorbid doses of infection or his subclinical attack and so is immune to all save perhaps the most massive doses of infection

It is clear that in a modern city any infective agent unless completely novel will meet with considerable resistance which compensates in some degree for the greatly enhanced opportunities of transmission This is especially true of those common diseases following which a lifelong immunity results for example measles epidemics of which occur roughly every two years Here the virus must await a fresh increment

of susceptibles Those children who were under six months at the last epidemic and those who suffered only the mildest of subclinical attacks from which they derived but a transient immunity, plus those babies born in the interval will constitute the victims and as these are aggregated in day nurseries nursery and infant schools, the opportunities are obvious Considering the population as a herd it has been calculated that as soon as a certain percentage becomes susceptible an epidemic becomes an immediate possibility The fact that epidemics do not always occur when they are due according to the statisticians shows however, that there is more in it than simple percentages

The general nutrition and environment of the population must next be considered History shows that great wars are almost always followed by great plagues Not only because of the disorganized sanitation and of the widespread movements of population so it is said but because the general level of nutrition is depressed below the danger point and the under nourished population falls an easy prey to organisms whose virulence is exalted by every easy passage This seems only rational and certainly in circumstances of widespread malnutrition such diseases a tuberculosis spread rapidly but the application is by no means universal The pandemic of influenza in 1918 one of the major plagues in history attacked not the old and enfeebled and the needy but the young and vigorous and the well to do Again in spite of widespread malnutrition due to the depression many cities in the United States succeeded in almost abolishing diphtheria (at any rate temporarily)

by immunization Speaking generally however there can be no doubt that the level of nutrition and the level of severity of attack are closely related and that furthermore once the level of nutrition has been depressed below a certain point the mechanism of the body s defence against attack seems to fail entirely

As a final major factor we must consider the **weather** the influence of which is common knowledge In summer flies and other insect vectors abound to pollute our food and organisms however introduced will multiply readily in foodstuffs unless refrigeration is adequate—hence food poisoning dysentery and gastro enteritis And though the snow and sleet of winter may not conjure infection from the soil nor the east wind blow it over the sea yet they chill us devitalize us and drive us indoors to huddle over our fires and incidentally to effect a mutual exchange of upper respiratory bacterial flora at a temperature highly favourable to their proliferation It is interesting to note that a person who sleeps out of doors seldom catches cold whereas anyone who undertakes a continental railway journey in winter (in superheated trains with tightly closed windows) seldom fails to do so

In the actual course of an epidemic a complex mechanism is released which may perhaps be summarized as follows Of those first exposed the wholly susceptible will succumb at once The organism invigorated by a favourable passage will now proceed to attack not only further wholly susceptibles (with the production of increasingly severe attacks) but some partially immunes as well This process will continue until the epidemic reaches its height By

that time many substantially immunes will have had trivial attacks and very many more will have had subclinical attacks the additional immunity derived from which will protect them at least until the end of the epidemic This will be brought about by the increasing hostility of the environment depressing the infectivity and virulence of the infective agent and by the rapidly dwindling number of susceptibles affording increasingly fewer opportunities of transmission The issue may then be determined by a change in the weather or some other event inimical to propagation of the organism and the epidemic comes to an end

Principles and Practice of Preventive Isolation

Since infectious disease results from the successful invasion of susceptibles by specific infective agents prevention may function either (*a*) by reducing the number of susceptibles or (*b*) by controlling the sources of infection In practice both methods are employed the former especially on an increasingly wider scale—witness vaccination against smallpox and immunization active or passive against enteric diphtheria tetanus measles and so on Where the sources of infection are water supplies food sewage and the like control is a well established and highly efficient function of the Public Health authorities But where the source of infection is the human body and especially the respiratory tract control is vastly more difficult since many of these diseases

are most infectious during the stage of invasion before their true nature is recognized and many people are unsuspected carriers For this reason isolation of the sufferers in special hospitals though undoubtedly of great value has not proved so efficacious in prevention as was anticipated except in those conditions such as smallpox enteric etc where the patient remains more or less uniformly infectious throughout the course of the disease These hospitals are however becoming of increasing value in treatment As they harbour patients suffering from a great variety of different infections it is obvious that spread of one infection to a patient suffering from another (*cross infection*) must at all costs be avoided Cross infection does unavoidably occur from time to time but considering the possibilities is relatively negligible To achieve such results certain administrative principles and nursing methods are essential these we must now examine

We must first recognize that all diseases are not equally infectious As noted in the section on epidemiology this depends on the size of the infecting agent the site of the local lesion and the character of the disease Note that the most infectious site the respiratory tract is the one invariably favoured by the most infectious agent—the virus Note also that a condition in which the agent escapes to the surface of the body in discharges is much more dangerous than a clean one Finally there is the biological phase in which the agent happens to be at the moment (see p 48) and the resistance of the host This last has special significance in hospital A case of streptococcal pneumonia in an adult fracture ward may

be negligible as a source of infection to others but in a ward of young children debilitated and vulnerable following measles or whooping cough might prove a menace

As the result of experience we may classify the common infections in roughly the following order of infectivity —(a) **diseases of high infectivity** spread by contact droplet and possibly air borne—*smallpox chickenpox measles* some forms of *enteritis* early *whooping cough influenza* and the *common cold* (b) **diseases of moderate infectivity** spread by contact and droplet—*rubella mumps late whooping cough* cases of *scarlet fever* and *diphtheria* with *discharges* and (depending on the circumstances) *pneumonia* (c) **diseases of low infectivity**—*uncomplicated scarlet fever* and *diphtheria erysipelas* the remaining forms of *enteritis enteric* the *dysenteries* and *puerperal fever* Distinctions will of course have to be made thus puerperal scarlet fever is normally spread by contact yet if an attendant nurse should come to harbour the organism in her nasopharynx spread will be by droplet in her case and the disease will become correspondingly more infective Again cerebro spinal fever (meningococcal meningitis) is spread by droplet seldom by frank clinical cases but either by carriers or by those undergoing subclinical attacks Frank clinical cases might thus be regarded as minor risks though it is usual to segregate them

Spread of infection is prevented primarily by allocating patients with any one given infection to an open ward devoted to that infection alone The staff of the ward is special to it and whenever a nurse is changed to a ward dealing with a different infection

she bathes washes her hair and makes a complete change of clothing before taking up her new duties It often happens however that a patient requires admission who is suffering from or has been exposed to more than one infection or from a condition so rare that there is no common ward devoted to it or that a patient admitted with one infection has been unknown to the hospital authorities incubating a second and subsequently develops it It is clear that in such cases special measures will have to be taken to limit spread Those in common use are described as barrier bed isolation cubicle and chamber nursing

Barrier and **Bed Isolation** nursing are practically identical In either case the patient his bed locker and belongings are presumably surrounded by an imaginary barrier They are to be regarded as untouchables and a red card is usually displayed to signify the fact Three gowns hang by the bedside one for the maid one for the nurse and one for the medical officer Before doing anything to or for the patient the nurse washes her hands puts on a gown and mask and when finished removes the gown and mask and scrubs the hands especially the finger nails under which infection is prone to linger Each temperature chart has its own pencil the patient is allocated his own thermometer face cloth comb brushes and so on Bed isolation and barrier nursing are quite adequate for the diseases of low infectivity grouped as *c* —and probably also for those moderately infective conditions in group *b* but they are inadequate for group *a* At the same time it is surprising what generous bed spacing and

rigorous attention to the rules can effect Of these, the most important is the thorough *washing of the hands* in running water for preference since the most important disseminator of infection is the attendant s finger The bowl of antiseptic often proudly displayed is worse than useless since in the dilutions employed even when freshly made, a nurse would need to soak her hands for ten minutes to secure satisfactory sterilization Hospital authorities should therefore provide abundant handbasins hot water clean towels and an appropriate hand lotion in such wards since it is useless to expect attendants to wash conscientiously (especially in cold weather) if the water is cold the soap harsh and the towels are already damp from repeated use Not only do the resultant chapped hands preclude satisfactory washing thereby defeating the whole purpose of the ward the unclean towels may easily become reservoirs of infection Care should also be taken not to load such wards too heavily with cases requiring very frequent or elaborate attention experience shows that above a certain pressure the system inevitably breaks down

Bed spacing in infectious hospitals should provide a minimum of eight feet between bed centres but ten or even twelve foot intervals are desirable The latter is the standard in the London County Council Infectious Hospitals Something similar is obviously necessary in all children s hospitals the greater the bed spacing the less cross infection is likely to occur While such spacing is not so essential in adult wards the overcrowding so common in many general hospitals is deplorable and makes one wonder what would happen if a really virulent infection were to gain a

footing These things move slowly It is only a century since a British physician travelling in France remarked how much superior the hospitals of Lyons were to those of Paris since in Lyons he seldom saw more than two patients to a bed whereas in Paris there were sometimes anything up to six ! Perhaps the physician of the future will regard our modern open and especially our general hospital wards with much of the horror and amazement with which we contemplate the simplicity and ignorance of our predecessors

The large open ward is undoubtedly falling into disrepute The great disadvantage of the hospital treatment of the infectious diseases is that in spite of the specialist skill and knowledge available the patient sharing as he does a ward with perhaps nineteen others is exposed to any infection which any one of the others may develop and the ward is at a standstill until the quarantine has expired For this reason *chamber* nursing is coming more and more into favour

Cubicle and **Chamber** nursing aim at the complete segregation of the patient He is surrounded not by imaginary but by real barriers which in the case of cubicles extend for seven feet in height on three sides (the fourth side being formed by the wall) whereas in chambers the partitions extend to the ceiling These partitions are composed largely of glass to permit of observation In cubicle wards there is therefore a common air space above the level of the partitions through which the more active droplet and air borne infections can pass from patient to patient and in practice they are not reliable where those highly

infectious diseases in group (*a*) are concerned though said to be adequate for (*b*) In chambers, however the patient is completely enclosed and any infection can be nursed in them especially when they communicate freely with the open air Cubicles are therefore seldom constructed nowadays chambers being the rule though (unfortunately) often loosely miscalled ' cubicles

The technique of cubicle and chamber nursing is in essence identical with that of barrier and bed isolation Opinions differ as to the best arrangement of chambers Some are built in double rows with a central enclosed passage between them others in a single row giving on to a verandah wholly or partially open to the air The former tend to be regarded with disfavour the latter are undoubtedly excellent Again the siting of the wash basins and the type of door fastening are both matters of importance The wash basin must be *within* the chamber otherwise the door handles may readily become contaminated Of course with outside basins swing doors can be used but these are not really satisfactory as they tend to blow open on windy days if the windows are as they ought to be kept open Needless to say adequate washing facilities in chambers are essential

On admission to an infectious hospital a patient with a single disease is allocated to the appropriate ward Should infectious complications be present he is sent to a bed isolation ward or to a chamber depending on the disease similarly with patients suffering from or exposed to more than one infection whether a recognized fever or an infectious skin disease e g scabies Cases or contacts of group (*a*)

infections must go to chambers cases or contacts of group (*b*) may go to a barrier ward at a pinch but preferably to a chamber cases or contacts of group (*c*) may well go to a barrier ward or to the ordinary ward on bed isolation' This last must not be overdone however When cross infection arises in a ward the first measure is to isolate the culprit It is also prudent to isolate for a time all children admitted from residential schools institutions and indeed other hospitals since they often display an amazing capacity for incubating a rich assortment of infections

A generous allowance of isolation accommodation is therefore essential in the modern acute infectious hospital in fact it has been calculated that one half of the total accommodation should consist of chambers Before the war hospital authorities everywhere were busily providing such facilities Furthermore experiments were being made in breaking up ordinary open wards into units of four beds separated by screens seven feet high, each unit with its own wash basin so as to diminish the risk of spread and reduce the number of contacts should cross infection occur The diagram (Fig 7) of a ward at the North Eastern Hospital London shows a modern approach to infectious hospital architecture

Isolation in a private house is often required A secluded well ventilated quiet room as near as possible to a bathroom should be chosen It is customary to hang a sheet redolent of carbolic outside the door This is unnecessary but it is a useful bogey to frighten busybodies away The technique is that of barrier nursing Current and terminal disinfection may have to be carried out if the Local Authority

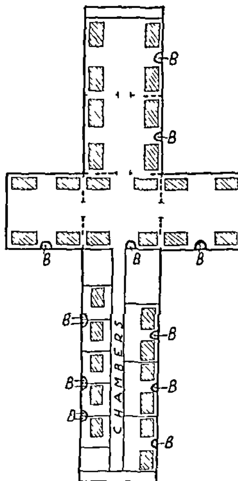


Fig 7—Modern Ward at the North Eastern Hosp tal London The broken lines represent 7 foot screens partially dividing the ward into five 4-bed units Note the generous isolat on accom modation and the abundant washing facil tie (B=wash bas n) [By kind permission of the London County Council]

insists—which it rarely does nowadays except in cases of smallpox

Disinfection is termed *current* when it is carried out during the course of a disease and *terminal* when it is carried out at the termination **Current disinfection** involves the sterilization by boiling or steam of all crockery utensils and instruments that have come into contact with the patient and the burning of all swabs and dressings and pieces of gauze or lint used as handkerchiefs Sputum is received into mugs containing disinfectant which are later autoclaved Bed wear and linen is soaked in disinfectant and wrung out before being sent to the laundry This is essential in smallpox chickenpox and the intestinal infections In these latter used bed pans and urine bottles should have disinfectant added and be allowed to stand for a time before being emptied into the sluice

Terminal disinfection is carried out when a patient is discharged He bathes washes his hair and puts on clean clothing The discarded clothing the mattress pillows and blankets are sterilized by steam the underwear shirts and linen being soaked in disinfectant and sent to the laundry If he is discharged from a chamber the bedstead is swabbed down with disinfectant the walls are washed the floor is scrubbed and the windows are opened widely before admitting a fresh case

When a patient has been nursed in a private house the terminal disinfection of books mattresses and other articles too valuable to be burnt and likely to be spoiled by soaking may have to be undertaken by the local health authority Formerly it was customary

to pull all furniture away from the walls open all drawers, close the doors and windows and spray liberally with disinfectant After six hours the room was reopened The walls were then stripped and washed with hot lime before being repapered Although still recommended, these drastic procedures are seldom carried out unless the walls badly require attention or following a case of smallpox

The Health of the Attendants —The attendants as a source of infection is a subject usually discreetly avoided and it is indeed an embarrassing one Nevertheless it is quite clear that while a medical officer or a nurse would hardly go on duty with a notifiable infectious disease they may do so with a sore throat or a severe cold This is quite wrong Sore throats are very infectious as of course are colds, and such an infection communicated to a child already debilitated will not only reproduce itself but may lead on to mastoiditis or even pneumonia Again attendants on children with nasopharyngeal infections speedily pick up the organisms themselves and if conditions in the nose or nasopharynx are unhealthy may readily become carriers not only of the C diphtheriae but of the streptococcus One has known a medical officer who whenever afflicted with a cold almost certainly gave rise to a number of cases of scarlet fever in various wards and nurses whose trails were similarly marked Nurses indeed are far more likely to infect patients than doctors since they come into much closer and more frequent contact with them than do the latter and besides are prone to pick up fondle and kiss young children and babies This really ought to be strictly forbidden Furthermore,

although circumstances do not always permit us to remain away from our work when ill we can at least dispense with all but the most essential examinations requiring close contact and we can also wear a mask Nurses can do likewise If this were common practice there would be less inexplicable morbidity among our patients especially the children

SECTION IV

EXAMINATION AND TREATMENT OF FEVER PATIENTS

BEFORE making any examination the student must elicit the history This is essential in arriving at a diagnosis and especially to avoid such elementary mistakes as the mis diagnosing an attenuated measles as rubella It is customary to warn the history taker against asking leading questions lest the patient s imagination be thereby stimulated but unless he is a person of unusual leisure the student must accept that risk In any event since so many patients are children he will often have to get his history at second hand from the mother if not indeed at third hand from a nurse who has interviewed her

The first enquiry to make is what previous illnesses has the patient suffered from ? Next the history of the present illness When was the patient last perfectly well ? *When did he refuse his food and is he* reluctant to swallow ? If so since when ? What other symptoms are there and when did they appear ? e g headache vomiting diarrhœa rigors pains in the back limbs or joints cough (whether simple spasmodic or croupy ?), swollen glands aural or nasal discharge and rash with site and date of first appearance Has the patient had serum previously or *any other injections* ? If so what for and when ? Is he or anyone in the family allergic ? Has he been exposed to any other infection ? Lastly where does

he come from? If from home has anyone in the house street school or clinic been admitted to or discharged from an infectious hospital recently? If he is from another hospital a convalescent home or a residential school he should arouse suspicion that while exhibiting one infection he may be incubating another

The patient must now be examined And here a word of warning to the student What distinguishes the good physician from the bad in nine cases out of ten is not superior knowledge but *the habit of making a careful examination* After all the vast majority of the patients which the doctor sees are suffering from common conditions with the signs and symptoms of which after a reasonable experience he becomes quite familiar Where he goes wrong is in not taking the history properly but more commonly in not eliciting the signs Beware therefore of the spot diagnosis and the slipshod examination Three of four of the mistakes in diagnosis that one sees in hospital are due not to want of knowledge but to *failure to examine* A surprisingly large number are even due to neglect of the first essential—to *look* at the part of which complaint is made! This may seem hard to believe but nevertheless it is a well established and sorry fact

Inspection can teach us a lot First of all note the decubitus which may often afford a clue Does the patient lie on his side with head retracted as in cerebro spinal fever or persistently on the affected side as in unilateral pneumonia or pleurisy? Does he shun the light as in the photophobia of measles or meningitis? Are his knees drawn up as in colic or acute abdominal

mischief? Is he flushed and febrile or pale or cyanosed? Is his expression anxious and worn or vacant as in the very toxic or is it frowning as with severe headache or constant irritation or pain? How does he react when spoken to? Is he alert, or mentally dull, as in toxæmia or semiconscious or comatose? Does he speak clearly or nasally and thickly,' as in quinsy or severe inflammation of the throat or tremulously and with an effort as in severe toxæmia? Even this brief examination should in many cases suffice to tell the physician whether his patient is gravely ill or no and the student should perfect himself in subconsciously appraising the minutiæ of expression and behaviour in disease Is the patient miserable as in measles quiet listless and docile as in diphtheria or unconcerned as in rubella? The student should now note the frequency tension, and rhythm of the pulse and also the respirations—are they normal or rapid and shallow as in pneumonia or noisy and laboured as in laryngeal obstruction? Does the patient cough? If he does is his cough loose croupy or spasmodic or does the patient restrain it because of the pain it evokes—as in pleurisy or the acute abdomen?

The student must next inspect the whole body in the best light available Ignoring for the moment any rash that may be present let him begin at the scalp Are there any pustules or scab of impetigo to be seen or felt or any thinned out patches of hair as in *tinea capitis*? Impetigo again is common on the face in children The face is often puffy and bloated in measles and whooping cough and markedly so in smallpox It is the most common site of ery

sipelas and as already mentioned readily indicates fever or cyanosis The circumoral pallor of scarlet fever and the malar flush of pneumonia should be recognized at sight and the pear shape of salivary mumps is characteristic

Turning to the individual features the **eyes** often show a typical salmon pink injection in rubella The lids are often puffy in whooping cough or nephritis and puffy inflamed and crusted with discharge in measles For future reference always note if a squint is present Next the **nose** Crusted excoriated nostrils with thick scanty sanious nasal discharge are characteristic of anterior nasal diphtheria a profuse watery or purulent rhinorrhœa is seen in acute infections—measles severe nasopharyngeal diphtheria or scarlet fever A *unilateral* foul smelling discharge suggests a foreign body In severe toxæmia the **lips** are dry and cracked and often crusted with sordes Herpes simplex may accompany any sharp upper respiratory tract infection but is often most profuse in cerebro spinal fever The lips may often give information of value In acute streptococcal throats they are a bright red In cyanosis they are mauve or even blue nowadays this may be due as often to the sulphonamides as to oxygen lack Finally do not forget the **ears** Otorrhœa readily occurs in any nasopharyngeal inflammation most commonly perhaps in scarlet fever measles or tonsilitis

Inspection of the neck, trunk and limbs must follow The tonsillar **glands** enlarge in tonsillitis scarlet fever and diphtheria as well as in glandular fever tuberculosis the leukæmias Hodgkin s disease etc The posterior cervical group are nearly always

enlarged in rubella and glandular fever, and the axillary may be very important in the latter The submaxillary and the sublingual salivary glands may become enlarged and hard in stomatitis and mumps, almost the only adenopathy in which they participate

On the skin generally, pitting or scars may be seen The 'foveated' scars of vaccination are usually on the arm Smallpox pitting is profuse on the face chickenpox scars are to be seen on the trunk Scars *of acne may confuse they occur on the face and upper* trunk especially the back Papules and pustules of acne may be seen in the same areas Papules are common on the inner thighs and buttocks of children (urine rash) Should papules or pustules be present search should be made for a *vesicle* Vesicles occur ordinarily in chickenpox or smallpox and also in urticaria herpes and sometimes impetigo Are there any scratch marks ? If so the webs of the fingers and toes must be inspected for *scabies* which however may be obscured by a secondary impetigo or eczema and moreover may flourish anywhere on the delicate skin of young children A scabbed eruption most profuse on the back of the wrists and forearms under the breasts in the axillæ the groins and the genitalia would also suggest scabies of old standing Oval pink rings of ringworm must be carefully noted The buttocks in babies should be inspected for excoriation and the vulva in female children for vaginal discharge While much of the foregoing may seem medically trivial to the student he must remember that his notes on these points duly initialled constitute a legal document which may later on prove to be of first importance especially in hospital practice Infective

conditions are often alleged to have resulted from negligence on the part of the hospital staff paradoxically enough more often by parents whose children on admission are models of neglect than by those whose children show every evidence of care With the disquieting increase in the public appetite for litigation in matters medical there is need to take every precaution

During the foregoing inspection the student will have ample opportunity to examine any rash that is present we will now therefore discuss what he is likely to have seen

A **rash** or **exanthem** is a generalized usually transient alteration in the appearance of the skin due to bacterial toxins or other poisons In examining any rash the following points must be noted —

(1) *Is this a true exanthem?* Any rash due to generalized toxæmia should be present in some degree on all parts of the body and is usually present on the greater part of it An eruption on one limb or confined say to the lower abdomen does not constitute an exanthem as a rule As an exception the rash of enteric may be often very scanty

(2) **The site and date** of first appearance

(3) **The type** —Certain terms are used in description An *erythema* is a reddening of the skin (a blush is a transient erythema) A *punctum* is a point or dot A *macule* is a spot flush with the skin not palpable by the finger A *papule* is a raised macule and therefore palpable A *vesicle* (blister) is a transparent thin walled papule containing clear fluid a *pustule* is a vesicle (into which leucocytes have emigrated) and therefore yellow or opalescent *Petechiæ* are pinpoint

hæmorrhages at first dark red and later blue black as the blood is extravascular they do not disappear on pressure Flea bites are similar but show a central puncture mark, as they lie deeper they do not go blue *Ecchymoses* are larger extravasations of blood they usually appear crimson then purple then blue and finally green, as in the familiar "black eye A rash is described as *discrete* when the component elements are separate and *confluent* when they run together Combinations of these terms are commonly employed in description viz a maculo papule a discrete macular erythema Any rash but especially one consisting of vesicles papules or pustules is often referred to as an *eruption* or *outcrop*

(4) **The distribution** of the rash not only where present but where heaviest or absent Unless in smallpox or chickenpox acute infectious rashes are not well seen on the palms or soles because of the thickness of the epithelium though the essential mechanism is there at work—witness the subsequent desquamation Nearly all exanthemata have a recognized distribution to which they scrupulously adhere

(5) **The colour** may range from the vivid scarlet of typical scarlet fever through the brick red of measles to the brown of the later stages of the measles rash Rubella has a salmon pink tinge the dull red macules (rose spots) of enteric are well known

Prodromal rashes are especially common in virus diseases They may be erythematous or hæmorrhagic when the latter they often imply a heavy infection Such hæmorrhagic rashes are usually petechial but in fulminant infections ecchymoses may occur—e g

in smallpox when they may be accompanied by hæmorrhages from any or all of the mucous membranes Nose bleeding alone is not however a sign of grave vascular trauma as it may initiate any trivial infection in certain subjects

Heterogenous rashes may be caused by a variety of other agents which introduce or liberate some substance toxic perhaps only to certain individuals Notable are certain *foods drugs* or *garments* an *enema* or an injection of *serum* Of foods shell fish are notorious and canned foods especially canned fish have a similar reputation Pork is also a great offender in certain individuals indeed idiosyncrasy in this direction is proverbial Urticaria is the most common manifestation Drug rashes (aspirin salicylates bromides sulphonamides etc) may be morbilliform or scarlatiniform Flannel rash is usually a bright angry erythema corresponding to the area covered by a garment it is extremely itchy as is one form of napkin rash of babies which results from *the inadequate rinsing of napkins washed in strongly* alkaline soap Enema rashes may be coarsely morbilliform So called *septic* rashes may follow burns or wounds infected with hæmolytic streptococci when they are really varieties of scarlet fever in other cases they may present mixed morbilliform and hæmorrhagic elements of which the causative agent is unknown They are prone to occur about the knees and elbows

It may be noted here that rashes are still something of a mystery It is obvious that they represent changes in the blood vessels of the skin but why they should so constantly appear in the same place and at

the same time in the course of any given infectious process, why they should favour one area and avoid another, what governs their duration—these are just a few of the questions to which at present we can provide no answers in the vast majority of cases We have to take them for granted and learn their characteristics as carefully as we can

When confronted with a rash simulating perfectly that of some well known exanthem but with no other signs of the disease and nothing to account for their absence e g a typical measles rash with no fever no concomitant signs or symptoms and no history of the administration of a prophylactic injection, the student must go carefully into the foregoing possible sources of the eruption and he will frequently succeed in discovering the cause *No diagnosis must ever be made on a rash alone Whatever the rash may have suggested to him the student must next examine the mouth and throat* This must never be omitted There is hardly an acute infectious disease in which the buccal cavity has not some suggestive and often vital clue to offer Indeed a pale quiet oropharynx will promptly and conclusively refute the testimony of the skin in suspected measles or scarlet fever In the former Koplik s spots are often diagnostic in the latter the angina* and papillated tongue are essential In small pox or chickenpox lesions are often present on the palate or pharynx and may clinch a diagnosis hitherto uncertain In mumps the mouths of Stenson s and Wharton's ducts are often enlarged and injected The diagnosis of diphtheria can usually only be made

* This term is often confusing to the student *Angina* alone means an inflamed throat quite unrelated to *angina pectoris*

after the closest examination of the oropharynx Even in whooping cough a sublingual ulcer may afford confirmatory evidence not to mention that the exploration of the posterior tongue with a spatula will usually provoke a typical paroxysm in a genuine case

The patient should first be asked to put out the tongue (even quite small children will comply with this request) which very often affords valuable information though as a factor in diagnosis it has tended to become undeservedly neglected in recent years possibly because (thanks to the well known advertisement) it suggests a diagnosis arrived at on horseback in the public highway instead of in the laboratory It is by no means so unscientific as it would appear In nearly all virus infections the local lesion is in the nasopharynx and viruses as we have noted conduce to proliferation of streptococci Where streptococcal proliferation is proceeding a characteristic picture is produced The tongue is red and inflamed, and the papillæ are enlarged and prominent showing through a deposit of grey white fur (see Plate IV) Florid examples of this are seen in scarlet fever measles tonsillitis and so on but in mild degree it is extremely common in chickenpox rubella and often diphtheria Such a tongue indicates definitely that there is active infection in progress It is almost invariable in children when the above conditions hold good in adults it is often unaccountably absent A thick white uniform fur is the commonest appearance in the adult though in severe or prolonged infections it may appear as described or alternatively shiny raw and dry with a central brown deposit In

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observation and the young practitioner especially should be on his guard against plumping for a diagnosis when he is really uncertain merely because he is reluctant to admit his uncertainty Let him rest assured that he will damage his prestige far more seriously by rashness than by caution

TREATMENT AND MANAGEMENT OF THE FEBRILE STATE

There are certain general principles which govern the treatment of the febrile patient which in many conditions are the only measures we possess Briefly they may be defined as the reduction of his toxæmia the conservation of his strength and the securing of his maximum possible comfort The measures employed will be described now and to save repetition will be referred to when necessary later on as the general treatment of the febrile state

Water as we know is a prime necessity of life So long as we have an adequate supply of water we can withstand starvation for many weeks deprived of it our expectation of life is reduced to a matter of days The water content of the body is delicately adjusted to our activities muscular and secretory In fever the water balance is disturbed For reasons of its own the body retains all the water it can consequently the urine is diminished and sweating also ceases Even so the patient wants more water Water should be given liberally not only because the patient demands it but because it increases the secretion of urine and with the urine toxins may be

constipation a white fur is often evident although a white fur is not conclusive evidence of constipation The association of the 'raw beef' tongue with diabetes is of course well known

The student must next inspect the gums and mucous membrane of the cheeks where perhaps aphthæ, ulceration or Koplik s spots may be evident Finally he should depress the tongue and examine the uvula fauces tonsils and pharyngeal wall This should be done in all cases indeed in every sick child the throat should be examined The possibility of retro pharyngeal abscess should always be remembered The glands should next be palpated and in sharp or obscure infections the spleen felt for The heart must next be investigated The apex beat is first located and the rate and rhythm established The character of the sounds may next be noted a systolic *bruit* at the apex is common in febrile children and need not receive too much attention The chest is next examined and other systems are investigated as indications arise If the foregoing has been carefully carried out and the temperature ascertained the student should be in a position to suggest a diagnosis in the vast majority of cases

But not in all In quite a number of cases the signs are often equivocal and although a given diagnosis may seem most probable a doubt may linger in the mind In infectious work such doubts should always be respected otherwise one may do a lot of mischief such as passing a doubtful case of scarlet' into a scarlet fever ward only to find it covered with a measles rash the following morning The obvious thing to do with such a case is to isolate it under

reduced more often by the patient than the physician Indeed it is often difficult to persuade the febrile patient to take anything This is rarely of any moment A patient at complete rest in bed requires very little nourishment to keep him going and more harm is done by thrusting food on a reluctant patient than will ever result from a temporary fast

Where fever is prolonged and wasting rapid the situation is quite different but it will usually be found that such patients are quite ready to take food the only question that arises is what can they safely have ? While it is obvious that some care will have to be exercised there is no need for the extravagant and often senseless prohibitions in which our predecessors were wont to indulge and of which we ourselves are not altogether perhaps quite guiltless

In the acute stages **fluids** are pushed Glucose is valuable inasmuch as it is a potent source of energy readily soluble and not too sweet so that it can be tolerated in much larger quantities than sugar which though equally valuable is usually taken freely only by children Either may be added to lemonade tea or coffee Milk of course is a basic item either alone or incorporated in egg flips or in dilute milk puddings but it has been greatly overdone especially in the prolonged fevers such as enteric Some patients detest it and in any event after a week of milk diet even milk addicts may find it nauseating It should also be remembered that milk though a valuable food is a very good medium for the growth of micro organisms and is a common agent in the spread of epidemics if it is not pasteurized The possibility of its becoming infected *after* pasteurization

excreted Ice to suck water in tea coffee lemonade *or broth are all equally efficacious The patient should* be encouraged to drink by any and every means and it will often be found that, whereas he may refuse a drink in one form he will accept it readily in another Besides relieving thirst it keeps his mouth clean and a hot drink at night may with advantage replace a drug in securing sleep By contrast a chilled or iced drink may prove most acceptable to a patient whose throat is too painful to permit of his swallowing anything else

In the reduction of toxæmia apart from specific measures *water without* no less than water within is our most powerful ally Water without may take the form of a cold or tepid bath or a cold or tepid sponge Baths are not commonly employed in this country the cold and particularly the tepid sponge being preferred Sponging not only reduces hyper pyrexia it stimulates the circulation and encourages the kidneys to act It is one of the most effective ways of combating delirium and should be employed far more frequently than it is especially in view of the uncertain and often alarming results produced by drugs in that condition Sponging moreover cleanses the skin thereby diminishing the liability to bedsores and adds materially to the patient s subsequent comfort Furthermore it may be repeated as often as desired since there are no reactions and no contra indications

Diet in the febrile state is restricted to the more readily digestible articles In common with other secretions those of the stomach are diminished This is usually expressed in anorexia the intake being

infections provided that no undue draughts are set up Ventilation in general can hardly be considered here suffice it to say that it is not lack of oxygen but humidity and lack of air movement which are the great causes of discomfort Light good quality woollen blankets will conserve quite enough heat while allowing sufficient ventilation to carry away the layer of heated air next the skin and the surface moisture The patient should never be half cooked And yet this is a frequent tendency of both parents and nurses A child with a mackintosh below and an eiderdown or lumpy heavy blankets above must either throw off the bedclothes or wake up in a bath of sweat since there is practically no ventilation with such a combination Excess heat is a frequent cause of insomnia and the victim moreover is often wrongly described as febrile

The febrile patient is nearly always constipated This results from water conservation and whether it is at all harmful or not is uncertain In adults the psychological effect may be distressing but in children this is by no means apparent (Prisoners of war in Japanese hands on a starvation diet of polished rice often went for weeks without a bowel action and equally without any apparent ill effects) It is true that the bowel may act as an eliminator of toxins and for that reason should be encouraged *gently* to act On the other hand we do know that toxins present in the bowel may be reabsorbed if the contents are fluid at a point where they should normally be solid and that diarrhoea however produced has a pronounced enfeebling effect Hence the emphasis on the word *gently* The brisk purge at the outset

should not be ignored Milk should always be kept cool—i e at or below 40° C The addition of soda water gives it a pleasant detergent quality which is refreshing when the mouth is foul

Fresh eggs beaten up raw, or lightly boiled are very useful Soup and prepared meat juices with a little bread are often a welcome variation Ice cream with its high fat and sugar content can be very nutritious a staple article in America it is surprising that it is not more widely employed in this country As regards alcohol opinions differ The physician must balance its temporary (physically and psychologically) stimulating effects and its subsequent value as a hypnotic against the exacerbation of any existent gastritis which it may produce it has no specific curative value rather the reverse In all these matters the physician must be guided largely by the patient It is a good plan to give him no more than he asks for and conversely whatever he asks for to give always remembering that what is one man s meat is another man s poison Only water should be pushed We must remember that our knowledge of scientific dietetics is still rudimentary and that in cases of doubt the patient is least likely to be wrong

In convalescence latitude and liberality should be practised Red meat and fat fish (which require some hours digestion) are best omitted until the tem perature has been settled for some days otherwise a convalescent patient can have anything he desires

Fresh air is an essential of fever therapy Best of all is the open air provided the patient be kept warm with frequently renewed hot bottles The open window ranks next especially in acute respiratory

all a sleeping patient must never be disturbed unless for something vital no therapy can secure more comfort and rest than is expressed in sleep Sleep must be secured at all costs Insomnia is often due to cold or excess heat or to a noise or to the need of a urine bottle Commonly it is due to indigestion Sponging a hot bottle a hot drink or an alkaline powder may often suffice Should no such obviously remediable cause be found the physician must resort to drugs He should always enquire and note the amount and type of sleep his patients have secured

Those (hypertoxic) patients who are stuporous delirious or comatose will require special measures They are unable to indicate their needs so that regular opportunities for excretion must be afforded them and incontinence being common they may require frequent cleansing Bedsores easily develop these must be avoided by scrupulous attention to the pressure points Further unless the toilet of the mouth is frequently and thoroughly carried out parotid or submaxillary abscess inhalation pneumonia or an intractable gastro-enteritis may ensue These are rarely seen nowadays but they are not mythical and were once fairly common

The stuporous patient is easily managed but the delirious patient is a problem He almost always attempts to get out of bed Restraint is easy while he is still prone once sitting up he may require forcible measures If possible it is better to guide his movements to a harmless termination than to oppose them directly Should he persist mechanical restraint must be applied Two bed sheets one passing over

—a persistent refrain even in modern textbooks—is clearly a piece of superstition a relic of what used to be called the 'lowering regimen' wherein any plausible form of torture was legitimate If physicians sampled their own remedies they would not supplement the miseries of an already anxious and toxic patient with a pharmacological entero colitis Aperients employed should be of the mildest character and probably an enema is safest of all Any purgative which excites a free and fluid flow is bound to exacerbate the existing dehydration, for which nature promptly retaliates with a bout of constipation

The temperature pulse and respirations must be recorded twice daily and in severe cases four hourly The number of motions per day should also be noted, and in babies the weight should be registered once a week The urine should be examined daily where albuminuria is discovered in females a catheter specimen should be obtained to exclude vulval contamination A daily bed bath is essential, with additional spongings as required The *toilet of the mouth* is one of the essential measures and must on no account be omitted The cheeks gums tongue and hard and soft palates are swabbed out with lemon and water glycothymolin or glycerin and tannic acid after which the lips are cleansed and smeared with an emollient non poisonous application such as vaseline Attention is best given at regular intervals it is then less likely to be forgotten The attendants must strike a balance between fussiness and neglect the first need of the febrile patient is rest and too much attention in which he is never given a moment to himself is almost worse than none at all Above

stimulation of the heart eucortone or pituitrin may be of service in raising the peripheral tension

No measures can have any success however while the root cause the toxæmia remains unchecked If no specific serum is available to combat this and no chemotherapeutic agent to check the organisms from whence it derives the patient must rely on his own defences An intravenous saline with or without glucose may be of help and striking success may often result from a transfusion even a small one Should this fail to reactivate the defence the physician is usually at the end of his resources He must learn to resist the formidable if often silent pressure of relatives and others to do *something* no matter what when there is no indication that anything he may do will be of benefit Such therapy—for appearance sake—is more likely to prove mischievous than helpful In medicine judgment is still essential that is to say it remains an art An injudicious dose of pituitrin for example has before now produced a fatal hæmorrhage Every therapeutic measure must be judiciously assessed beforehand Unless it is clear that the patient is likely to be the gainer on balance he is better left alone Nature after all knows what she is about whereas often the physician if challenged can adduce no more than a pious hope in defence of his procedures It is more than likely that in the years to come many of our measures will appear almost as grotesque and absurd as the agonies to which Charles II was subjected on his deathbed Let this wholesome reflection temper our zeal

Prognosis in the acute fevers in general will depend on a number of factors which may be briefly

him knotted to a second which passes under the bed can be so adjusted as to allow him sufficient play for his energies without permitting him to escape Sponging must never be omitted Of drugs hyoscine luminal or paraldehyde per rectum are best Morphia is bad

The stuporous patient requires nasal or rectal feeding and the catheter must be regularly employed A drop of olive (or castor) oil should be applied to the corneæ from time to time since the normal cleansing and lubricant action of the lids is in abeyance, and the epithelium if dessicated may readily exfoliate and thus provide the starting point for an ulcer Caution is also very necessary in the use of hot water bottles since burns all too readily occur

The patient *in extremis* whether conscious or not is succumbing to his toxæmia He often lies limply with half closed lids and cooling limbs as the circulation slowly fails It is customary to resort primarily to heart stimulants but there are two fallacies in this In the first place his heart muscle possesses so much reserve and no more Like a balance at the bank this can be squandered or eked out to the last penny every time we administer a stimulant we draw a cheque on the cardiac reserve In the second place unless sometimes in diphtheria death is due not so much to failure of the *heart muscle* as of the *circulation* This is due to loss of contractility of the peripheral arterioles producing a condition analogous to shock where the patient bleeds into his own capillary lake Maintenance of the body heat by hot bottles or the electric cradle is much more likely to succeed than

incubation period so that the contact has either developed the disease or has escaped with certainty by the time the quarantine period has expired The segregation period is the length of time a patient *suffering from* the disease requires to be isolated e g Rubella quarantine period twenty one days segregation period—until rash has faded—at most seven days In most *virus* infections the segregation period should *antedate* the first characteristic sign of the disease by some days Thus in measles the patient is *most infectious* during the initial catarrh which may occasionally precede the true rash by a week

Sore Throat in General

Of all complaints that the student will encounter (and himself suffer from) during his infectious work sore throat is probably the commonest This is quite understandable if we reflect for a moment All infection as we have stressed must effect an entry into the body somehow This it can only do through the *broken* skin or through one or other of the channels by which the body communicates with the outside world These the rectum vagina and urethra—apart from venereal or puerperal disease—may for all practical purposes be dismissed as potential gateways of infection We are left therefore with the nose and mouth opening into the pharynx—the oro naso pharynx—as the main portal by which infection can effect an entry

The oro naso pharynx is in fact the main route by which the vast bulk of infectious diseases gain access

summarized as follows —(*a*) the promptness with which specific treatment (where it is available) is carried out e g serum in diphtheria (*b*) the age of the patient every year below ten and over sixty renders prognosis less favourable (*c*) the general nutrition of the patient—the lean and over plump are both handicapped the former in all infections but especially in diphtheria the latter in certain con ditions notably pneumonia (*d*) the presence of

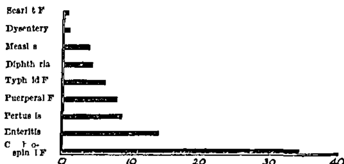


Fig 8—Death rate per 100 cases in L C C hospitals 1932–37 *

another pre existent or coincident disease thus measles superadded to active pulmonary tuberculosis may produce a rapidly fatal exacerbation while the coincidence of measles or whooping cough with one another or with diphtheria may result in a very unpromising situation

The quarantine period of any infection is the time for which a person *exposed* to the disease requires to be isolated before mingling again with his fellows It is usually one day longer than the longest known

* Before the intro luction of the more pow ful sulphonamides

incubation period so that the 'contact' has either developed the disease or has escaped with certainty by the time the quarantine period has expired. The segregation period is the length of time a patient *suffering from* the disease requires to be isolated, e.g. Rubella: quarantine period twenty-one days; segregation period—until rash has faded—at most seven days. In most *virus* infections the segregation period should *antedate* the first characteristic sign of the disease by some days. Thus in measles the patient is *most infectious* during the initial catarrh, which may occasionally precede the true rash by a week.

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to the body Infected dust may be inhaled infected droplets may impinge directly on the oro naso pharynx where they may proliferate or they may be inhaled or swallowed infectious material may be introduced by the finger or by an infected article such as a handkerchief spoon or pencil And of course infected food or drinks may in the ordinary course of events be unsuspectingly introduced and swallowed

The reaction of the oro naso pharynx to the introduction or lodgment of such infection will depend largely on the nature of the infecting agent Thus in the case of the *Eberthella typhi* or the dysentery bacilli which merely pass through in food there is no obvious local reaction at all at any time Nor as a rule does the tubercle bacillus at first entry produce any reaction to speak of though it may initiate a cold But in the case of those bacteria normally adapted to flourish in the upper respiratory tract e g the streptococcus the pneumococcus the meningo coccus and the staphylococcus as well as almost all the pathogenic viruses a reaction will follow—not immediately—but in due course depending on the incubation period of the invading infective agent This reaction may range from a slightly dry throat with or without a little nasal discharge to a severe cold or may even go as far as a florid and extremely painful tonsillitis Constitutional symptoms —i e chills fevers vomiting and so on may be slight or absent in the first and second instances but will probably be severe in the last Mild reactions are especially liable to follow infections in persons who already possess a high degree of immunity to the

infecting agent thus a nurse in a scarlet fever ward with a high general resistance to streptococci may experience merely the mildest reaction from invasion by a streptococcus of dangerous virulence which if communicated to a patient devoid of such immunity might produce a violent attack of scarlet fever

In general then a cold or a sore throat is a danger signal announcing that some sort of infection has quite recently been sustained and that the stage of invasion has now arrived In a child it may signify the onset of measles mumps rubella chickenpox smallpox influenza or infantile paralysis and of course of scarlet fever or diphtheria or it may merely indicate an attack by one of those indefinite (and probable multiple) viruses that give rise to what we call a common cold In a nurse it indicates that she has received a dose of infection of one sort or another which of course she may develop but to which she may be more or less immune and which therefore she may often shake off in a few days without incident But—and this is important—she may very well transmit such an infection in the course of her duties to others who are not immune with dire results

A glance at Fig 9 will readily show what opportunities for exploitation are open to an agent which establishes itself in the oro naso pharynx Thus it can spread straight down into the larynx trachea and lungs or up the Eustachian tubes to the middle ears and mastoids or into the maxillary antra or any or all of the sinuses—frontal ethmoidal or sphenoidal It can also spread directly down the œsophagus causing œsophagitis but in practice it seldom does What is

far more likely to happen is that the infective agent is swallowed in the sputum and thus introduced into the stomach and intestines perhaps to set up inflammation there In addition to any or all of these extensions it *may pass into the blood and this it habitually does in* the case of pneumococcal pneumonia meningococcal meningitis and probably in most if not all virus diseases *Sore throat therefore may constitute the* prelude to a wide range of possible and often perilous eventualities It is the first signal that infection has gained a footing and that battle has been joined It must never be underestimated or ignored

Treatment —*In purely* virus *sore throat there is* usually little more than a feeling of rawness or dryness Treatment is hardly required and in any case is in effective *Coccal proliferation is to be feared* however it will be marked by the intensification of fever and of local injection and usually by the appearance of exudate To guard against it it is tempting to employ sulphonamides or penicillin prophylactically, especially *penicillin lozenges A warning may be introduced* here however because it is likely that these anti biotics may be employed intermittently for very brief periods and in very small dosages thus possibly encouraging the appearance of sulphonamide or *penicillin resistant forms If they are to be employed* let them be employed decisively otherwise the last state may be worse than the first

SECTION V

THE STREPTOCOCCAL INFECTIONS

MANY attempts have been made to classify the streptococci but none is completely satisfactory They have been classified on morphological grounds (e g *s longus brevis*) or by their association with a certain habitat or a certain disease (*s salivarius s erysipelatis*) or by their fermentative reactions with sugars Nowadays they are commonly divided by their behaviour when grown on a blood containing medium Either the blood is unaltered (non hæmolytic) or it shows a greenish yellow colour due to incomplete hæmolysis (α hæmolysis) or finally it shows a clear decolourized zone of complete hæmolysis (β hæmolysis)

We are here concerned only with the last—the β hæmolytic streptococci Of these some thirty different serological types have been recognized by agglutination tests about twenty five of which give rise to scarlet fever From this it follows that some thirty different streptococcal antigenic complexes may confront the body defences of which about twenty five carry in addition a variable amount of an erythrogenic or rash producing fraction (which may also vary slightly) We may therefore regard each individual streptococcus as carrying a sheaf of antigens Suppose the complete range available to be A B C D E F G one given strain might possess them all another A B C D E F another perhaps

A, B C, D E G and so on while another might possess only A B or A C It is clear therefore that an infection with an A B organism would result in little or no immunity to an organism consisting mainly of C D E, or F which goes far to explain many of the anomalies hitherto observable For it is common experience that while immunity to the erythrogenic fraction of the toxin is fairly readily developed immunity to streptococcal infections as a whole is uncertain and transient Thus (as a result of reinfection with a different type) secondary strepto coccal tonsillitis is common in convalescence from scarlet fever though a rash (true relapse) is rare But the erythrogenic fraction of the toxin though clinically so assertive is relatively unimportant inasmuch as the virulence of any given strain of streptococcus is not to be measured by the intensity of the rash it produces and a case of tonsillitis due to a non erythrogenic strain may be just as dangerous and infectious as the most florid scarlet fever

Of the scarlet fever encountered in London some 60 per cent of cases are due to the first four types Since type 4 contains relatively few and weak antigens the immunity it produces is feeble indeed even the erythrogenic fraction is so poorly represented that a reinfection with a virulent polyvalent strain (rich in antigens) practically approximates to an invasion by a different organism Hence in type 4 cases relapses with rash production are common and the relapse is usually much more severe than the original attack By contrast the ' Dochez (type 10) strain from which Dick toxin and the therapeutic serum are made, is a virulent strain so polyvalent

that its antiserum is valid in almost every scarlatinal infection

The Dick test is based on the fact that the toxin injected intradermally will in a susceptible produce a localized erythema at the injection site In practice 0 2 c c of a $\frac{1}{1000}$ dilution is introduced usually on the left arm a control with heat inactivated toxin is done on the right An area of erythema, the size of a sixpence or a shilling will develop in susceptibles The result should be read in about 24 hours as fading is rapid The test is a good indication of immunity to such antigens as are carried by the Dochez strain and especially to the erythrogenic fraction

The Schultz Charlton test depends on the power of the therapeutic serum to counteract the erythrogenic action of the toxin 0 2 c c of the former is injected where the rash is heaviest and most confluent This is usually on the abdomen The test should not be done where the rash is blotchy—e g the limbs A positive result is indicated by an area of blanching the size of a halfpenny or a penny which appears in about twelve hours The test if done within the first two days is quite reliable

It will be clear from the foregoing that should an infection with an erythrogenic hæmolytic streptococcus occur elsewhere in the body other than the oropharynx a scarlet fever rash may well result This is the genesis of **surgical** and **puerperal** scarlet fever and apart from the unusual situation of the local lesion these conditions do not differ materially from the ordinary disease

The streptococci implicated in erysipelas and many cases of puerperal fever unmarked by rashes are

also fully hæmolytic In the latter an anærobic variety has frequently been isolated No doubt many instances of these diseases are also due to scarlatinal organisms, but since adults are largely immune to the erythrogenic fraction no rashes develop Cases of these diseases however do not react so favourably to scarlatinal serum as does scarlet fever proper possibly because it is primarily an antitoxic and not a bactericidal serum and the course of these diseases is governed more by proliferation of the organisms rather than by toxæmia Drugs of the sulphanilamide group or penicillin are therefore much more likely to prove effective than serum

Scarlet Fever

Scarlet Fever is an acute specific infectious disease characterized by inflammation of the throat and mouth and by a typical rash

Cause—as noted the *streptococcus hæmolyticus scarlatinæ* The organism is found in the nose throat and all discharges throughout the disease and frequently for long periods thereafter should unhealthy conditions obtain Carriers are common though often intermittent Thus the organism may persist in an infected antrum or sinus and only over flow when some intercurrent infection (e g a cold) causes a flare up

Incidence—any age Commonest between the fifth and tenth years Second attacks are by no means rare

Incubation Period —Two to seven days

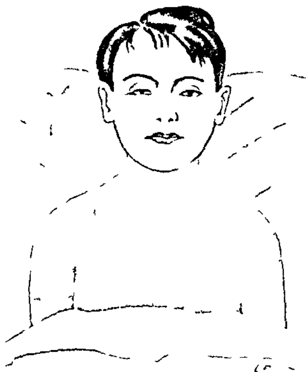


PLATE III

Scarlet Fever.—The rash. Note the flushed face and the circumoral pallor. The rash is more marked about flexures and tends to be coarser and blotchy on the arm. The punctation ought to be a little finer.

Facing p. 95

Segregation Period —Usually three weeks or until all discharges have ceased Desquamation is *not* infectious and should be ignored

Pathology —The basic element is the proliferation of the organisms in the oropharynx producing toxins which when absorbed result in the fever and (by dilatation of the superficial capillaries) the rash Unless in certain rare hypertoxic and perhaps septic cases the organism does not invade the blood The blood shows at first a polynucleosis and later an eosinophilia

In the presence of serum the sulphonamides and penicillin death from the uncomplicated mild type of scarlet fever prevalent in this country to-day is almost unknown In former days when toxic and septic cases were prevalent post mortem examination might reveal in toxic cases the changes customary in septicæmia—intense congestion of all the viscera and probably hæmorrhages into the skin and under the serous and mucous membranes In septic cases there would be intense congestion and usually ulceration of the fauces enormous swelling of the cervical glands perhaps with ulceration of the larynx and a septic pneumonia Pus in the sinuses middle ears etc would also be encountered

Course —The onset is abrupt with fever vomiting and sore throat The latter becomes progressively worse and within twenty four hours the enanthem or mucous membrane rash is well developed (see Plate II) This consists of a bright injection of the buccal mucosa generally most marked on the fauces and tonsils The latter may be swollen and covered with exudate The palate—hard and soft—may be stippled with punctations foreshadowing the skin

rash yet to appear The lips are bright red and the tongue is covered with a thick fur except at the edges where the enlarged inflamed papillæ show through In a further twelve to twenty four hours the exanthem (skin rash) appears (see Plate IV) This produces a typical picture The face is deeply flushed excepting an oval area surrounding (and contrasting with) the bright red lips—the *circumoral pallor* The rash proper begins on the neck and spreads rapidly over the trunk and limbs It consists of a background of brightly flushed skin against which the enlarged capillary loops in the papillæ show up as a stippling of myriads of tiny points of a deeper red—hence the description a *punctate* erythema On the limbs these punctations are much coarser Often many tiny sudamina may be seen *At the flexures especially in* front of the elbow extravasation from the loops may occur and petechiæ develop when present this is known as **Pastia s** sign Because of the thickness of the epidermis, the rash is seldom seen on the palms or soles

In a sharp case untreated by serum this picture may persist for a week or more Defervescence is by lysis As the rash fades desquamation begins usually about the end of the first week It commences on the neck continues on the trunk and may persist on the palms and soles for six weeks Desquamation con sists in the shedding of the horny layer especially over the papillæ consequently the most typical variety is the pinhole often best seen on the fingers resembling a pinhole made in paper looked at from the reverse side On the hands and feet the dead skin may come away like a ragged glove exposing a red



PLATE IX

The t ngue n S a l t F v r— e l tr w berry

F i g p 97

shiny area of new epithelium beneath *The scarlet fever (streptococcal)* tongue is at first heavily furred with enlarged inflamed papillae showing through at the margins—the *white strawberry* tongue (see Plate III) Later it clears from the edges inwards leaving a film of fur in the middle and a few scattered islands of white among the prominent papillæ at the margins—the *red strawberry* tongue Finally as the inflammation subsides it clears completely and acquires a purplish tint—the *raspberry* tongue This picture though best seen in scarlet fever is not exclusive to it and may be encountered wherever hæmolytic streptococci are active in the buccal cavity—e g often in measles diphtheria chickenpox or rubella It is not usually typical in adults

Types of Scarlet Fever—Four types are usually described —(1) the **simple type** described above (2) the **septic** type (*scarlatina anginosa*) in which there may be a fulminant infection of the tonsils perhaps with the formation of a streptococcal membrane or with severe ulceration perforation or even destruction of the soft palate *Extension* to the larynx may occur and gross cervical adenitis and profuse nasal and aural discharges may be present from the outset (3) the **toxic** type (*scarlatina maligna*) Since all cases of scarlet fever are of necessity toxic this name is misleading The term hypertoxic would be preferable where marked delirium prostration and circulatory failure are the dominant features of those extremely severe cases which undoubtedly (though rarely) occur the term toxic scarlet fever has however been applied to illnesses characterized by slight or moderate angina profound toxæmia

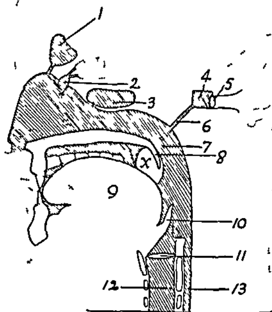


Fig. 9—Connections of the upper respiratory tract 1 Frontal sinus 2 Lachrymal sacs and ducts 3 Sphenoidal sinus 4 Middle ear 5 Tympanic membrane 6 Eustachian tube 7 Nasopharynx 8 Palate x Tonsil 9 Tongue 10 Epiglottis 11 Larynx 12 Trachea 13 Œsophagus

and a sparse petechial rash Such a picture might represent the onset of any overwhelming infection and one must often wonder how the diagnosis was arrived at unless with the eye of faith Much the same applies to (4) the hæmorrhagic type where bleeding from the mucous membranes petechiæ and ecchymoses with perhaps a transient scarlatiniform rash usher in a rapidly fatal intoxication Such cases could nowadays be verified by throat swabs blood cultures and the typing of the organisms involved In this country rapidly fatal cases of any type are now excessively rare the overwhelming majority being simple with an *occasional* septic and very rarely a truly hypertoxic example It is probable that in these last cases we are mostly dealing with a septicæmia

Complications occur in roughly the following order of frequency adenitis rhinitis otitis arthritis *sinusitis* nephritis endocarditis etc These may be grouped as *early*—occurring during the first ten days and *late* usually about the third week They may be again subdivided as *local* and *general* Local complications—adenitis rhinitis otitis and sinusitis —*result from* direct extension of the organism from the throat *whereas general* complications are usually due not to the organism *but to the toxin* Either may be late or early Nearly *always the onset of a* complication is signalized by a bout of fever which persists as long as the complication is advancing

Adenitis of the tonsillar glands occurs in some degree in almost every case The glands are moderately enlarged and tender but they usually subside in a few days Occasionally (especially in septic cases) the enlargement is considerable, the surrounding

cellular tissues of the neck are involved in a hard, tense, brawny swelling—"bull neck"—as in hypertoxic diphtheria (Fig 10 below) In such cases suppuration is *likely*, *even cellulitis of the neck may* ensue

Rhinorrhœa is also common *In septic cases it may*



Fig 10 — Bull neck in scarlet fever is much rarer than in diphtheria but may occur in septic cases

be profuse thin and sanious *later* becoming *purulent* It is often very persistent Late rhinorrhœa is most infectious and is an important *agent of spread* *No case with a moist nose should be discharged* Rhinorrhœa is also a *prime* source of *impetigo* Cases of persistent rhinorrhœa should be isolated

Arthritis (*toxic*) usually occurs about the end of the first week It is more common in adults It affects mainly the small joints of the hands and feet which may become slightly enlarged and tender They usually clear up readily In those rare septic cases however where the organism has penetrated into the blood *suppurative* arthritis of the larger joints may ensue

Sinusitis is not uncommon While any sinus may be involved the ethmoid seems to be most frequently picked out Where this occurs there is persistent fever headache and probably œdema of the lids though sometimes the condition is latent Pus dormant in a sinus may prove an active source of infection months later

Nephritis —Albuminuria is common in scarlet fever n all febrile states but nephritis is nowadays vely rare It occurs not during or following utial fever but during the third week as a or localized hæmorrhagic glomerulo nephritis et may be insidious with increasing albumin and diminishing excretion of urine or it may be sudden with sharp fever and a drastically reduced output of urine containing albumin blood and casts There may even be suppression Concomitantly there is often recurrent angina with adenitis The vast majority of these cases terminate favourably in one two or more weeks but rarely the condition advances steadily marked by headache vomiting increasing arterial tension anæmia and progressive emaciation to terminate in anasarca uræmia and death

Endocarditis —Although so frequently and confidently ascribed to scarlet fever by cardiological

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In regard to nephritis we may believe either that the initial toxæmia injures the glomeruli in such a way that the damage is not manifest for about three weeks or that it merely sensitizes them so that should a reinfection occur an allergic flare up may ensue Certainly the latter theory seems the more plausible and would also seem to apply to endo carditis when it occurs

departments, endocarditis, at any rate during the course of the disease, is rare Many children manifest a systolic bruit during the initial fever which soon disappears The belief that the rheumatic type of carditis is commonly *initiated* by scarlet fever is in my own experience quite unfounded though an existing lesion may be reactivated Further when endocarditis does arise *de novo* it is a late and possibly 'allergic' phenomenon (*vide infra*) which may as readily attack the aortic as the mitral valve and is not necessarily associated with any signs of rheumatism

Pericarditis Pneumonia, and Empyema are likewise extremely rare While almost any lesion may occur in septicæmic cases in the usual case chest complications are so rare that when they occur one may justifiably suspect some deviation from normal pathology Nothing is indeed more striking than the fact that though hæmolytic streptococci are proliferating abundantly on the tonsils in the scarlet fever of to day they seldom pass down even as far as the larynx whereas in measles although initially scanty they readily invade the respiratory tract This irresistibly suggests the conclusion that unless previously devitalized by a virus or other invader the respiratory mucosa offers a formidable barrier to the hæmolytic streptococcus

Pathology of Late Complications —As previously mentioned adenitis otitis rhinorrhœa etc may either occur during the first week or during the third This fact has naturally aroused much speculation as to the pathology of the late complications Recent work however has gone far to offer an explanation

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Rare Complications —Erythema nodosum and purpura may appear

Diagnosis —This is usually easy In the acute stage it is made on the presence of both the enanthem and exanthem *It must not be made in the absence of either* As regards the rash concentrate on the trunk it may be equivocal on the limbs and remember that it should be uniform if blotchy, suspect it As regards the enanthem typical tongues are rare in adults but there is always angina As confirmatory tests (*a*) the Schultz Charlton is almost always positive in an *early* rash but it must be performed where it is heaviest and most uniform—usually on the abdomen (*b*) the Dick test is usually *positive* for four or five days it usually swings to *negative* at the end of a fortnight and (*c*) a swab for hæmolytic streptococci should give numerous colonies A negative Schultz a negative Dick or absence of hæmolytic streptococci will throw serious doubt on the diagnosis In cases deferred for observation pinhole ' desquamation of the hands in three weeks is a useful sign though not infallible In doubtful cases either the positive Schultz or the Dick swinging from positive to negative are the only conclusive signs Culture for hæmolytic streptococci is helpful

Differential diagnosis —This is often given from tonsillitis, diphtheria measles rubella etc In typical examples of these diseases there should be no confusion the clinical pictures are quite different The first two show no exanthem But the second day rash of rubella and the *prodromal* rashes of measles, smallpox and especially chickenpox may

cause considerable difficulty So too at times may serum food and drug rashes The differentiation is given in the table (pp 109 110) Every case suggesting scarlet fever during a measles epidemic must be rigidly scrutinized

An occasional mistake is to confuse exfoliative dermatitis But here the erythema and desquamation are seen together There is no enanthem Transient heat rashes in children in hot weather are similarly devoid of enanthem or indeed of any sign of morbidity

Prognosis —The death rate of scarlet fever is nowadays less than 0 5 per cent This is due not only to improved therapy but to an undoubted decline in virulence especially during the past twenty years Scarlet fever is to day a mild disease But as this is said to have occurred periodically in the past we must not assume that it will be permanent In any event septic and toxic cases are now extremely rare and death from scarlet fever uncomplicated by mastoiditis or nephritis is very uncommon

Treatment falls naturally into two parts The funda mental cause of the condition is the proliferation of the organisms in the throat These may therefore be attacked by means of the sulphonamides or penicillin But this may not affect the toxæmic manifestations—the fever rash and so on—for a couple of days If toxæmia is severe then the antitoxic serum may be employed when the temperature will fall and the rash will fade Serum alone however will not usually check the organisms so that otitis etc may occur in spite of it It is therefore logical to employ chemo therapy ordinarily and to include serum whenever conditions demand it This is sometimes estimated

in terms of pyrexia so that as a rule of thumb any patient with a temperature of 100 F or over on the day after admission is given serum Many clinicians however prefer to leave the body to develop its own immunity in mild cases and therefore omit sulphonamides unless in sharp infections

Apart from the above treatment is that of the febrile state Special attention is paid to the urine a daily specimen being examined albumin occurring after *defervescence and apart from serum sickness being* regarded with suspicion If albuminuria occurs in female patients a catheter specimen should be examined The diet ranges from fluids to 'light' and full within about a week the belief that a prematurely generous diet might induce nephritis has now been generally abandoned

It is usual to keep patients in bed for ten days or a fortnight and to discharge them in three or four weeks provided they are well and free from all discharges These periods may have to be extended in severe cases

Treatment of complications is on general lines *Adenitis* if suppurative should not be incised too early *Arthritis* yields readily to aspirin as a rule unless suppurative when drainage is required *Mastoiditis* in scarlet fever is not usually such an urgent problem as in measles or influenza Rest adequate fluids and a bland diet curtailing protein (and salt should œdema occur) usually suffice for *nephritis* One complication often ignored *rhinitis* may be dealt with at length This is common not only in scarlet fever but in measles acute nasopharyngeal and chronic anterior nasal diphtheria In the first three

it is usually thin and watery becoming later purulent in the latter it is usually thick and often sanious If unilateral and foul *smelling* suspect a foreign body Rhinitis is one of the *commonest* causes of impetigo the child inserting his finger into his nose and then abrading and infecting his skin with the infected finger In female children vulvo vaginitis may occur in similar fashion The eye a cut or a ruptured vesicle may be likewise infected Such rhinitis should always be cultured Streptococcus scarlatinæ or C diphtheriæ is often present when sulphonamides or diphtheria antitoxin may be required If persistent the cause perhaps an adenoid pad or a foreign body must be removed The nostrils and upper lip should be frequently cleaned and anointed with some non poisonous application such as vaseline and finally splints should be applied to prevent auto infection

PROPHYLAXIS OF SCARLET FEVER

Attempts to immunize susceptibles along the same lines as diphtheria have been made for many years Small but increasing doses of scarlet fever antigen (dilute toxin or toxoid) are *injected* at weekly intervals If correctly graduated immunity to the erythrogenic fraction at least should result The procedure is not very satisfactory because since the toxin is not pathogenic to laboratory animals each batch has to be standardized by assessing the skin test dose (i e the amount required to produce a positive Dick reaction) on human volunteers This method is

unreliable Consequently if too little toxin be given no immunity will result if too much a "miniature" attack of scarlet fever may ensue—i e mild fever with a transient punctate erythema but without angina

Patients exposed to scarlet fever can be protected for two to three weeks by an injection of scarlet fever antitoxic serum About 2 000 to 3 000 units are required the amount (in bulk) of the serum required will depend on the degree of concentration They may also be protected by a course of sulphonamides In either case they should be safeguarded by removing the source of infection

CONTROL OF OUTBREAKS

If a case of scarlet fever occurs in a childrens ward (1) isolate the patient (2) quarantine the ward —usually for a week (3) look for and remove the source if possible—some child with either an active tonsillitis or with rhinorrhœa otorrhœa or impetigo or possibly a septic wound Remember it may be one of the staff—with a transient nasopharyngeal infection masquerading as a cold If the source can be removed no more need be done if not, nose and throat swabs for hæmolytic streptococci from all patients should be examined though even if these are present they are unlikely to be the source unless there is a discharge or catarrh Dick tests should be done all round this will divide the patients (at any rate approximately) into susceptibles and immunes All susceptibles may then be given an appropriate dose

DIAGNOSIS OF A SCARLATINIFORM RASH

Cause	*Scarlet Fever*	*Rubella*	*Prodromal Measles*	*Prodromal Chickenpox*	*Prodromal Smallpox*	*Food Drug or Enema*	*Serum*
Type	Punctate	Finely macular	May be punctate blotchy	May be punctate blotchy	May be punctate blotchy	Blotchy not punctate	Blotchy some urticaria
Colour	Scarlet	Pink	Scarlet brown tinge	Scarlet (very rare)	Scarlet	Scarlet or deeper red brown	Scarlet
Distribution	Trunk and limbs.	Face trunk and limbs	Usually trunk.	Usually trunk	Extensor surfaces or bathing drawers	General.	Patchy
Pyrexia	+ +	±	+ +	±	+ +	−	±
Angina	+ +	−	∓	∓	±	−	±

operations or wounds about the nose and face Formerly common in surgical and lying in wards it has now been almost abolished by aseptic technique

Incubation Period —Uncertain Probably two to five days

Segregation Period —None usually enforced While formerly erysipelas may have spread—from suppurating wounds—rapidly round a ward in these days of aseptic technique such spread should be out of the question As far as one can see most cases arise by the patient s infecting himself from his nose or from a discharging ear, and spread from case to case in a ward is in one s own experience unknown Elaborate isolation precautions are quite unnecessary and the condition could easily be dealt with in general hospitals at a pinch

Signs Symptoms and Course —The onset is sudden with shivers and sharp fever though a dramatic invasion with rigors vomiting and even delirium is not unknown In about twenty four hours the affected area of skin—often the bridge of the nose—becomes tense red swollen and shiny Very soon it begins to spread in one or other direction and as it does so the spreading edge evolves This is the most swollen part of all and if the fingertip be drawn lightly across the normal skin next in line of invasion this edge can be felt abruptly demarcating the zone of inflammation (see Plate V) Blebs are often a prominent feature though they may be absent As the edge advances the skin behind it gradually *returns to normal* often showing a slight branny desquamation In uncontrolled cases the process may extend fanwise to involve the eyelids forehead and

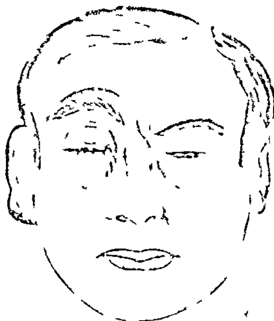


PLATE V

[illegible]

scalp though the commonest extension is on to the cheeks producing a butterfly patch Spread in some cases may be very marked thus commencing in one pinna the erysipelas may traverse the face to involve the other and then return again or it may invade the scalp or lower face or even migrate to the trunk or limbs Gross but temporary disfigurement may thus ensue In infants the navel is often the initial focus While actual pain is seldom complained of there is an uncomfortable feeling of tenseness pricking and burning in addition to the febrile reaction which as noted may be exceedingly sharp

Rarely the process extends to the mucous membranes of the mouth and throat As a result *laryngitis œdema of the glottis* or *inhalation pneumonia* may ensue But more commonly a fulminant infection usually in the aged may penetrate to the subcutaneous tissues especially of the scalp producing a cellulitis with greatly enhanced fever and a corresponding deterioration in the general condition In fatal cases the typhoid state may rapidly supervene

More usually the condition runs its course in about a week though it may continue to wander about the skin for a month or more **Relapse** used to be extremely common and often proved worse than the initial attack **Recurrence** is also to be feared It is not unusual to encounter patients who have suffered repeated attacks

Complications —**Adenitis** of neighbouring glands **cellulitis** and abscesses may occur **Gangrene** is rare so too are **nephritis** and **pneumonia** Should **septicæmia** ensue pneumonia is almost certain to be

one of the multiple manifestations of this extension of the infection

Diagnosis is easy in most cases *Herpes ophthalmicus* is a source of error but the distribution the absence of a spreading edge the typical vesicles and the (usually) severe pain should easily differentiate it *Eczema solar* dermatitis and the dermatitis following *burns* as well as the allergic reactions to *insect bites* are also confused *Cellulitis* is another pitfall though in varicose ulceration cellulitis and erysipelas may co exist Blondes of plethoric appearance if afflicted with rosacea may arouse unfounded suspicion

Prognosis and Treatment—The prognosis is excellent Erysipelas responds more promptly to the sulphonamides than perhaps any other disease They should be given in full doses for two or three days In addition the general therapy of the febrile state should be instituted and soothing local applications such as calamine cream are grateful though with sulphonamide therapy the necessity for such measures is short lived It is also well to try to establish the source from which the patient has infected himself such as the nose an infected lachrymal sac a running ear or the like and warn him against the practice of infecting his finger and then rubbing or scratching himself and thus producing a relapse

Control of Outbreaks—These are nowadays very rare Should a case occur in a general ward it should be bed isolated Immediate attempts to identify the source should be made and special attention should be directed to possible sources in the patient himself Any patient with a purulent discharge should also be segregated The practice of bundling septic cases

indiscriminately into a ward unless under the strictest aseptic technique should never be countenanced

PUERPERAL PYREXIA

(While this condition is by no means invariably the result of infection by the hæmolytic streptococcus yet so many of the fatal cases are due to that organism that it has been considered proper to insert it here in order to emphasise such a malignant relationship Since a volume might well be written on the subject the student must realize that what follows is merely a brief summary of a protean condition)

According to the latest regulations a woman may be certified **puerperal pyrexia** if at any time within twenty one days of the termination of preg nancy there is a rise in temperature of 2 F or more sustained for or recurring within twenty four hours Note that while this definition rightly permits mor bidity following abortion and miscarriage as well as following full term labour to be notified it allows of any other febrile disturbance such as influenza being notified as puerperal pyrexia should it occur during the specified period The incidence of post abortion sepsis has shown a marked increase during recent years

Cause —Infection implanted on the puerperal perin eum vagina cervix or uterus is the primary cause This may be **endogenous** when the organisms are already present as with a pre-existing gonorrhœa or it may be **exogenous**—that is introduced from elsewhere either in the patient herself (as when

B coli spread along a complete tear from the rectum or the patient auto infects herself from a rhinitis or the like) or from an outside source, through the medium of her attendants Attendants may infect the patient directly by droplet from their naso pharyngeal secretions or by first contaminating their fingers or instruments with these secretions the results are the same Many authorities consider droplet to be much the commonest mode of spread The identity of organisms in the dying patient's blood with those of the attendant s nasopharynx has more than once been established The organisms con cerned are commonly (a) ærobes *hæmolytic strepto coccus staphylococcus aureus B coli* and the *non hæmolytic streptococci*, (b) anærobes *B Welchii non hæmolytic anærobic streptococcus* and *B pseudo necrophorus*

Incubation Period—Very variable—two days to two weeks

Segregation Period—Patients should be removed from labour wards and maternity units and rigor ously isolated until all discharges have ceased

Pathology—The normal skin and mucous membrane as we know possess vigorous powers of defence But when their integrity has been destroyed by tearing crushing and the rupture of their nutrient blood vessels they may become excellent culture media for organisms Following labour the perineum is frequently torn The vaginal mucous membrane may be ruptured torn off or devitalized by crushing exposing the relatively defenceless tissues beneath Frequently the cervix is lacerated Blood clot another excellent culture medium is abundant in the

uterus and genital canal Organisms introduced may proliferate without hindrance they may ascend through the vagina to the cervix and uterus with great rapidity They may invade directly the cellular tissue or the peritoneum of the pelvis at any stage they may penetrate to the thrombosed blood vessels this danger is particularly acute at the placental site

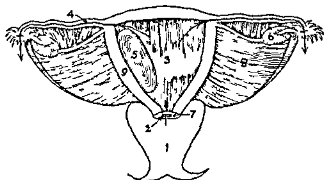


Fig 11 —Path of infection in Puerperal Sepsis Note (5) the placenta and (9) the placental site one of the chief danger points Spread to the broad ligament (8) is easily achieved but infection via the Fallopian tube (4) is rare unless in gonococcal cases

where access to the enormously dilated pelvic veins is readily effected Conditions for invasion are so favourable that the wonder is not that so many cases occur but that so many patients escape The prime factor is the virulent invader One has seen the most extensive lacerations the result of prolonged and exhausting labour followed by little or no morbidity *per contra* small and insignificant traumata have resulted in a fatal septicæmia The reputation of

abortion in this connection is especially sinister here anaerobes are often involved

Morbid Anatomy—This will depend on the course the condition has pursued An almost invariable finding is a subinvoluted uterus showing a necrotic endometritis especially at the placental site The uterine wall may tear like wet blotting paper When death as commonly has been due to septicæmia the heart muscle is often equally flabby and the spleen diffluent There may be general peritonitis, pneumonia lung abscess or empyema Purulent pericarditis is not very rare Multiple abscesses in the liver spleen and kidneys and in the more protracted cases even in the muscles may often be found. In the pelvis apart from peritonitis or cellulitis the veins of the broad ligaments are not infrequently thrombosed in abortion cases abscesses in the broad ligaments may result from infection conveyed by a perforating instrument

Varieties—Cases may be grouped as (1) local *genital tract infection*—uterine cervical or vaginal (2) *pelvic inflammation*—this comprises pelvic cellulitis *pelvic abscess* pelvic peritonitis and tubo-ovarian abscess (3) general peritonitis (4) metastatic conditions pyæmia and septicæmia Other manifestations are (5) venous thrombosis ("white leg") (6) breast abscess (7) cystitis (8) pyelitis and (9) puerperal insanity

Course—For the sake of convenience we may imagine that the first five varieties follow one another in orderly sequence as indeed they may occasionally do, with the proviso *that the process may be self limited or arrested* by treatment at any stage When

a fulminant infection supervenes, the earlier stages are likely to be extremely brief or absent

Genital tract infection includes the so called sapræmia believed to be due to retained products These may of course be present but it is very doubtful if they can genuinely give rise to fever About three in four of all cases notified show some sepsis of the uterus cervix vagina or perinæum which remains local and uncomplicated and from which normal recovery is made Of the remaining 25 per cent a certain number go on to pelvic inflammation such as **pelvic cellulitis** Here a hard moderately tender swelling may be felt low down against the pelvic brim on one or both sides When the inflammation involves the broad ligaments to produce **pelvic peritonitis** there is pain tenderness on pressure and some muscular guarding in the iliac fossæ The patient may vomit and is sharply ill A **pelvic abscess** may form which will naturally require incision and drainage Should the process progress to **general peritonitis** the patient is desperately ill pallid anxious and in pain Fever is pronounced the pulse increases steadily in frequency the abdominal muscles become rigid and vomiting may be frequent and painful Distension may add materially to the patient s discomfort besides embarrassing the heart and diarrhœa which often supervenes is an ominous sign If due as usually to the hæmolytic streptococcus the outlook is extremely grave

Metastatic Conditions—*pyæmia* and *septicæmia* are merely variations upon the same theme Theoretically in pyæmia pus should be present in the blood whereas in septicæmia the organisms are constantly in

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Course—For the sake of convenience we may imagine that the first five varieties follow one another in orderly sequence as indeed they may occasionally do with the proviso that the process may be self limited or arrested by treatment at any stage When

It must be emphasized that all combinations and degrees of severity can occur Thus pelvic cellulitis and pelvic or general peritonitis may co-exist with septicæmia A breast abscess a white leg (q v) or pyelitis may also coincide With organisms circulating freely almost any lesion may arise In the days before chemotherapy was introduced I have seen a case of septicæmia complicated by general peritonitis broncho pneumonia double empyema and a purulent pericarditis make a recovery

Venous Thrombosis (white leg) —This may occur alone or in association with the foregoing usually in B coli infections The femoral veins are almost always involved The whole limb becomes enormously swollen œdematous and pallid There is grave danger that a fragment of clot may break off and block some vital vessel Pulmonary embolism may thus occur the patient experiencing a sudden pain in the chest coupled with dyspnœa cyanosis and extreme anxiety Should the pulmonary artery be occluded immediate death will ensue if a smaller branch be affected a lung abscess will follow

Urinary Infections —Cystitis and pyelitis are not uncommon Again the B coli is the commonest cause and such urinary infections may occur with pyæmia or septicæmia or alone They do not differ materially from the cystitis and pyelitis encountered elsewhere

Breast abscess following an infected cracked nipple is a frequent cause of morbidity The whole or more usually a sector of the breast becomes intensely swollen congested and painful The patient is sharply ill Axillary adenitis often ensues. Suppuration is usual with extensive pus formation

circulation It seems extremely unlikely that the former ever occurs but the term may stand for a condition in which an intermittent low grade septicæmia occurs resulting in localized suppurative metastases as distinct from the diffuse and widespread complications of a heavy blood infection In the latter a positive blood culture should be obtainable at any time but not invariably in the former Clinically either condition may pursue a very variable course In pyæmia there may be long continued remittent or intermittent pyrexia with a frequent pulse foul lochia but nothing otherwise of note until—often following a rigor—metastasis occurs This is often a deep seated abscess Following this localization a variable interval may elapse before another appears This process may continue the patient becoming progressively more emaciated until either a chequered and stormy illness terminates in recovery or a metastasis in some vital organ often the lung proves fatal On the other hand septicæmia may set in some forty eight hours after delivery with swinging pyrexia a rapid thready pulse and frequent rigors The patient may be drowsy or stuporous but more often the mind is clear and as she may have no pain, the gravity of her illness may escape the inexperienced eye But the patient herself is tremulous and apprehensive certainly that fallacious feeling of well being (*euphoria*) often spoken of as a characteristic of this condition must be very rare Death may follow within a few days without any other sign but most commonly general peritonitis broncho pneumonia lung abscess purulent pericarditis or arthritis appear singly or in concert before the end

any sort of acute or chronic upper respiratory infection should be permitted to have anything to do with a woman in labour In any case, the attendants should always wear masks and sterile gloves Existing genital tract infections should be cleared up before labour occurs and the patient should be carefully warned against auto infection Needless to say the higher the standard of obstetric practice is raised the less likely is puerperal sepsis to occur

Treatment and Management —This must depend on the condition present Usually the patient is best in hospital where she should be strictly isolated As a routine a local examination is required provided the patient can stand it The attendants wash up etc as for an aseptic operation The patient is placed in the cross bed position or the lithotomy position if an operating table is available The perineum is cleansed and inspected for tears A sterile catheter is next passed and a specimen of urine is collected in a sterile bottle A speculum and retractor are now inserted and the vaginal walls and cervix examined A cervical swab is taken and it is usual to have at hand a sterile catheter as well as a 20 c c syringe containing sterile glycerin The catheter is passed well into the uterus This alone may have a most beneficial effect if there are retained products or the uterus is retroflexed but if there is much sepsis 20 c c of sterile glycerin may be slowly introduced and repeated daily A sterile pad is applied to the vulva and a specimen of blood is taken and forwarded together with the swab and urine for laboratory examination

Subsequent treatment will depend on the condition

Puerperal insanity may follow a mild or sharp infection It is usually preceded by *insomnia* and often constipation The patient develops hallucinations of *sight and sound and delusions frequently of* persecution She may suspect her husband or her attendants of a conspiracy to dispose of her, obstinately *refusing food and resisting attention* She ignores the baby She is often suicidal or homicidal Needless to say she must be kept under the strictest supervision

Puerperal Scarlet Fever—As *noted this differs from* ordinary scarlet fever in that the primary lesion lies in the urogenital canal and not in the tonsils But whereas in ordinary scarlet fever general *peritonitis for example never occurs*, and septicæmia is an extreme rarity in the puerperal variety such com*plications are always a possibility* The prognosis of puerperal scarlet fever is consequently very much more uncertain and hazardous than that of the ordinary disease since *it is usually governed by the gravity or* otherwise of the associated condition

Prophylaxis—Puerperal infection only *too often passes beyond the physician's control* No effort should therefore be spared to *prevent* it All expectant mothers should be carefully observed from the sixth month onward, so that abnormalities or faulty presentations may be corrected well beforehand In*struction in personal hygiene should be given defective* teeth should have attention and besides routine urinary *examinations* the blood should be investigated *for anæmia which is still* far too common Much difficult labour can likewise be avoided by *timely* induction *Aseptic delivery should be invariable* and no attendant (*whether nurse or physician*) with

puerperal peritonitis coupled with septicæmia is debatable In pyæmia and septicæmia apart from dealing with the metastases attempts must be made to sterilize the blood In hæmolytic streptococcal and in staphylococcal infections penicillin and the sulphonamides have immensely improved the prognosis and either—or both—should be given in full doses In the non hæmolytic streptococcal cases as well as in some anærobic infections response to these drugs may be very disappointing The position in this field however is far from static and new anti biotics may well alter the situation radically in the future

In urinary infections the sulphonamide should be administered together with large doses of alkalis (60 to 90 grains) but if reasonably prompt recovery does not ensue a full urological investigation should be made Mastitis can be prevented by scrupulous care of the nipples should it develop penicillin will usually prevent suppuration if given in sufficient dosage in good time

White leg requires the affected limb to be raised on pillows and immobilized by sandbags until it returns to normal This may take six weeks Thereafter the patient gets up by easy stages when an elastic bandage should be worn as swelling tends to return by evening *Puerperal mania* may often be success fully dealt with by keeping the patient continuously under hyoscine for three or four days but expert advice should be sought

Control of Outbreaks —Segregate the patient strictly Investigate other patients and staff for any infective foci and the latter particularly for nasopharyngeal

found If possible the patient is nursed in Fowler's position to promote drainage Air rings are undesirable The amount and character of the lochia should be noted *Any solid matter should be placed in water and teased out, in which case blood clot will break up readily, and the structure of any other substance can be defined* This may consist of fragments of the membranes or of the placenta, indicating retained products or of the fœtus in incomplete abortion In such cases evacuation of the uterus may have to be undertaken *The nipples should be examined for* cracks cleansed after feeding, and hardened by swab bing with spirit If the temperature exceeds 100° F it is best to withdraw the baby from the breast This may necessitate the use of the breast pump the giving of *œstrogens* or the application of a tight bandage to suppress the milk flow

It is nearly always necessary to deal with the anæmia present *This may require anything from a simple iron mixture to a transfusion depending on* the severity of the condition It is considered advisable that in puerperal cases a hæmoglobin level of 60 per cent or below *should be met by transfusion*

As long as the lochia is foul glycerin may be re peated *If the vagina or perineum is badly lacerated low pressure vaginal douches may be employed, though* they are less popular than formerly Operation for repair is *best postponed* In pelvic inflammatory *conditions as in general peritonitis the sulphonamides* and penicillin have proved of the greatest value where the organism is sensitive but if an abscess has formed drainage *is essential* The value of laparotomy in

SECTION VI

DIPHTHERIA

DIPHTHERIA is an acute infectious disease the result of successful invasion of the body by a virulent strain of the *Corynebacterium diphtheriæ* This organism may attack mucous membranes or skin Wherever it settles it kills the tissues *en masse* producing on mucous surfaces a pearly white false membrane and on the skin a blackened area of necrosis Moreover a diffusible exotoxin is liberated from the local lesion which passes into the blood and attaches to certain tissues for which it has a selective affinity notably nervous tissues and the muscle of the heart (Some toxin may also pass up via the perineural lymphatics of the local nerves to the cord and brain stem) Hence the complications which occur in well-defined and almost serial order over many weeks The toxin interrupts temporarily the function of the tissue involved Should this interruption be intense and widespread or should the tissues involved be vital (such as the heart or the nerves to the muscles of respiration) death may ensue Otherwise the intoxication passes off much like the effects of anæsthesia and the tissues return to normal Permanent damage is almost unknown

Bacteriology—The organism may exist in three phases indistinguishable microscopically when grown on Löffler's serum—the *gravis* or starch fermenting

abnormalities or catarrh *Swab any lesions found* and the noses and throats of all staff Segregate suspected patients and curtail activities of or temporarily suspend suspected staff Revise aseptic precautions

form the *intermediate* or *'barred'* starch negative type and the *mitis*—the remaining starch negative forms The barred or striped appearance, often with 'club' forms is developed only when grown on tryptic serum agar The gravis type is usually associated with severe attacks and is always virulent the barred form is (also) almost always virulent and as a rule gives rise to less severe attacks while the mitis usually occasions a mild infection with a predilection for the laryngeal and tracheal mucosa Many exceptions to the above generalization occur and this is not surprizing since one variety is said to change to another in culture, and therefore might conceivably do so in the body None of the true *C diph* ferment lactose but all ferment glucose These fermentation reactions are very useful inasmuch as they enable the many non virulent diphtheroids (often morphologically identical) to be sharply differentiated In the investigation of a suspected case of diphtheria a Loffler slope is inoculated with a swab from the source If organisms morphologically suspect are found they are plated out on tellurite tryptic serum agar containing $\frac{1}{1000}$ copper sulphate This inhibits other organisms whose colonies show up black whereas those of C diph display a black centre with a clear grey margin These are transplanted to tryptic serum agar after which they are again examined microscopically the barred forms should now be recognized without more ado In any case fermentation tubes are inoculated (see table page 129) Only the non virulent forms of mitis now remain to be excluded This is done by inoculating a guinea pig intradermally with a suspension of the organisms Virulent strains

produce a marked erythematous reaction within twenty four to forty-eight hours

	Glucose	Lactose	Starch	Tryptic serum agar	Virulence
Gravis	+	—	+	Appearance normal	+
Intermediate	+	—	—	barred	+
Mitis	+	—	—	n rmal	+ —
D phtheroi ls	+ —	+	—	normal	—

Pathology —The membrane consists of laminated fibrin entangling the necrotic surface cells of the epithelium and various blood cells In general it is loosely adherent below the larynx and firmly adherent in and above it except in the anterior nose Toxin absorption is greater in those regions where it is most adherent and where the tissues are most vascular *Post mortem* unless death has occurred during the florid stage little may be seen by the naked eye Microscopically there may be degenerative changes in the heart muscle and bundle of His and in the axons of the peripheral nerves often extending back to the nuclei The nucleus ambiguus may be involved Hæmorrhages and degeneration in the adrenal are often present In hæmorrhagic cases extravasation may be seen principally from and beneath the mucous and serous membranes

Incubation Period —Two to four days

Segregation Period —Until two or three cultures at weekly intervals are negative

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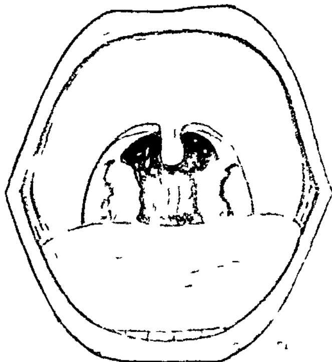


PLATE VI

Ton.illar D] l tl r ı —N to the me nbrane on e th r tonsil Apa t f om a on of nj tion at the bor ler of tl e me nbrane th tl r at s qu et

Fa ı j p I 9

munity The child is not at all ill The nostrils are excoriated fissured and often encrusted, and there is a little nasal discharge usually thick and often blood stained The adjacent skin of the upper lip is often crusted with impetigo a sign which in children should always arouse suspicion Occasionally a few shreds of membrane may be seen on the septum or lower turbinal The condition is static showing little tendency to spread posteriorly in contrast to naso pharyngeal diphtheria, in which spread forward from the posterior to the anterior nares readily occurs Why this should be is not clear The patients are often Schick positive and some can undoubtedly infect their open wounds with fatal results as one has seen happen Ordinarily as little toxin is absorbed from the anterior nose complications are rare but the child is a menace to others

Post nasal diphtheria alone is rare and confined mostly to infants and small children Only a little thick post nasal discharge may be seen and the diagnosis may be missed unless the student realizes what such a discharge may imply

Nasopharyngeal and tonsillar diphtheria will be described as one since the former includes the latter and the latter so often evolves into the former The diphtheritic process may commence anywhere between the middle turbinal and the larynx but it *almost always begins on one or other* **tonsil.** It may remain unilateral but in cases of severity spread occurs— to the opposite tonsil the faucial pillars the uvula palate and the nasopharynx proper depending on the sharpness of the infection and the stage at which the patient comes under treatment When the naso

Incidence —Any age but mostly children from five to ten years As cases given serum early in an attack may develop little immunity second attacks are not uncommon

Mode of Spread —By direct contact droplets fomites and the fingers of attendants occasionally by milk Carriers are common and dangerous

Varieties —Diphtheria may be classified first of all as respiratory and non respiratory Beginning at the nose **respiratory** diphtheria may be classified as (1) anterior nasal (2) posterior nasal (3) naso pharyngeal (4) tonsillar (5) laryngeal (6) tracheo-bronchial, and (7) diphtheritic broncho-pneumonia This classification is admittedly rather confusing but it does emphasize the point that severe diphtheria tends to involve not merely the tonsils but the posterior nares both aspects of the soft palate and the faucial pillars as well The description nasopharyngeal diphtheria is much more accurate than the old term faucial diphtheria which was formerly used to cover rapidly invasive and extensive infections equally with mild cases in which membrane was con fined to the tonsils *Tonsillar* diphtheria is the normal form and it is a matter of practical experience that when membrane spreads beyond the tonsils the case is likely to prove serious **Non-respiratory** diphtheria may be (7) conjunctival (8) aural (9) genital—vulval or preputial and (10) cutaneous Respiratory types are by far the commonest especially 1 3 4 and 5 Clinically the disease may be acute hypertoxic or chronic in the anterior nose

Course —**Chronic anterior nasal diphtheria** occurs usually in those with a high but not absolute im

The membrane may spread continuously the toxæmia become profound and the patient s condition steadily deteriorate He lies limply on his back the eyes half closed his expression one of exhaustion Though conscious and afebrile he is listless and quiet but subject to fits of restlessness Pallor is pronounced Because of the obstruction in his throat and nose his speech is thick and indistinct and he breathes stertorously through the open mouth He is docile and will submit readily to examination and seems to feel little pain in striking contrast to purely streptococcal infections A profuse watery sanious or mucopurulent discharge issues from the inflamed nares irritating the adjacent skin and thin tubes of membrane may present at the nostrils The cervical glands and the cellular tissues of the neck are involved in a hard prominent tense swelling obliterating the normal outlines—(bull neck)—an appearance suggesting and often tragically mistaken for mumps (see frontispiece) There is an intense and characteristic fœtor of itself diagnostic to the experienced The lips are cracked and dry and from the angles of the mouth blood stained saliva may trickle away A thin white margin of pallid skin outlines the mucous membrane of the upper lip—a characteristic sign which persists far into convalescence seldom seen apart from diphtheria

On examination the parts are seen to be grossly distorted The tonsils and uvula may be enlarged to double the tonsils meeting in the midline and pushing the uvula forward so as completely to occlude the faucial arch already narrowed and flattened by the œdematous soft palate and uvula All these structures

pharynx is involved a watery or purulent nasal discharge usually betrays the fact Spread downwards to the epiglottis larynx trachea and bronchioles may easily occur and if unchecked the disease may involve the whole respiratory tract Note that the lips tongue and œsophagus almost always escape

Signs Symptoms and Course—The disease begins *insidiously* The child is pale quiet and listless but makes no complaint of pain He may swallow normally and is slightly if at all febrile Indeed unless there is coincident streptococcal infection or the glands enlarge appreciably an unobservant mother may notice nothing amiss If the throat be inspected periodically the process will be seen to begin as small discrete translucent or gelatinous patches on one or other tonsil These gradually coalesce and become opaque to form a continuous firmly adherent glistening white membrane which cannot be torn off without bleeding and will not break up in water The membrane is bordered by a thin zone of injection and œdema in sharp cases but the oropharynx generally may be normal There may be slight enlargement of the tonsillar glands Should streptococcal activity coincide however the oropharynx may be brightly injected and the tongue be of the streptococcal type The glands will be rather more enlarged and in the early stages the face may be flushed

At any stage the patient s resistance may rise (or treatment intervene) to limit progress Many cases are self limited But in those whose resistance is very low or who have sustained an infection of extreme virulence the hypertoxic stage may develop

The membrane may spread continuously the toxæmia become profound and the patient s condition steadily deteriorate He lies limply on his back the eyes half closed his expression one of exhaustion Though conscious and afebrile he is listless and quiet but subject to fits of restlessness Pallor is pronounced Because of the obstruction in his throat and nose his speech is thick and indistinct and he breathes ster torously through the open mouth He is docile and will submit readily to examination and seems to feel little pain in striking contrast to purely streptococcal infections A profuse watery sanious or mucopurulent discharge issues from the inflamed nares irritating the adjacent skin and thin tubes of membrane may present at the nostrils The cervical glands and the cellular tissues of the neck are involved in a hard prominent tense swelling obliterating the normal outlines—(bull neck)—an appearance suggesting and often tragically mistaken for mumps (see frontis piece) There is an intense and characteristic fœtor of itself diagnostic to the experienced The lips are cracked and dry and from the angles of the mouth blood stained saliva may trickle away A thin white margin of pallid skin outlines the mucous membrane of the upper lip—a characteristic sign which persists far into convalescence seldom seen apart from diphtheria

On examination the parts are seen to be grossly distorted The tonsils and uvula may be enlarged to double the tonsils meeting in the midline and pushing the uvula forward so as completely to occlude the faucial arch already narrowed and flattened by the œdematous soft palate and uvula All these structures

may be covered with membrane the appearance of which will vary with the type of case and the stage of disease It may be ominously thin transparent and gelatinous in a rapidly advancing case later it may be thick white and glistening and later still it may be grey white or yellowish (see Plate VII) In any variety it may be blackened or bordered with dark crimson hæmorrhages Strings of mucous or mucoid secretion mixed with frothy saliva often obscure the view

Such a picture may commonly appear about the fourth or fifth day or earlier in fulminant cases Death from circulatory failure analogous to shock may forthwith ensue but usually this stage is survived Left untreated the membrane may persist for ten to twelve days commonly spreading down to involve the larynx trachea and bronchi While this is hap pening the oropharyngeal membrane may deliquesce or separate often with much hæmorrhage from the mouth and nose If still untreated and provided that the patient survives the membrane may re form here and elsewhere prior to the introduction of antitoxin the process often continued for weeks Usually within two to four days after the administra tion of serum the throat condition clears up and the affected parts heal unobtrusively Interest shifts to the condition of the circulation the heart and the nerves which constitute the subsequent chapters of the disease They may remain unaffected in which case there will be nothing more to relate and recovery may begin forthwith But this happens only in mild or very moderate cases in infections of any severity the heart and nerves are almost invariably involved Consequently the further progress of the patient is

the history of the complications and will be later resumed

Laryngeal Diphtheria (Croup) —The larynx may be involved primarily or secondarily to a nasopharyngeal infection Membrane forms on the swollen vocal cords thus blocking the airway Again the onset is insidious The first sign is usually hoarseness of voice followed by the characteristic croupy cough As obstruction increases the breathing becomes rapid and noisy on inspiration (**inspiratory stridor***) The child phonates on *inspiration*—much as in an audible sigh—but the voice is lost Later on the accessory muscles of inspiration are called into play—the sternomastoids and scalenes—and the respiratory effort becomes extreme so that the root of the neck and (in children) the sternum are indrawn with each inspiration—**recession** Still later the lips and cheeks become cyanosed from oxygen lack and the child panic stricken and desperate starts up from time to time fighting for breath and flinging himself about thereby exhausting himself further The strain on the heart is intense the pulse becomes weaker and begins to intermit and finally the respiratory efforts weaken *The cyanosis gives way to pallor* (*pallid cyanosis*) inspiration becomes jerky dwindling to a feeble platysmal gulp and expiration is prolonged The child lies inert Operation at this stage may prove too late as even if the airway is restored the heart may fail In cases of purely laryngeal diphtheria which survive however few complications occur as little toxin is absorbed from the larynx or below

* The student must learn to distinguish *stertor* due to *pharyngeal* obstruction from the *stridor* of *laryngeal* obstruction

it Note that adults and larger children may show few signs of distress until obstruction is severe

Diphtheritic bronchopneumonia is a sequel to the foregoing usually discoverable only after tracheotomy Almost always fatal cases have occurred in which the patient has coughed up a membranous cast of the bronchial tree usually through a tracheotomy *wound and subsequently recovered*

Conjunctival diphtheria is rare Membrane forms on the palpebral conjunctiva later invading the globe It may involve and destroy the cornea with resultant blindness

Genital diphtheria is equally rare Membrane may form on the vulva or prepuce The former may spread widely One case of each has come to the author s notice Both were recognized late and both died

Cutaneous diphtheria is also rare There is a black area of necrosis surrounded by an intense dusky red zone of injection The central slough separates exposing a deep ulcer which may extend down to the underlying bone Cutaneous diphtheria may complicate any wound or abrasion and is not uncommon in chickenpox Toxic absorption may be considerable and even death may follow

Complications may occur in all but mild cases of diphtheria The sharper the attack the more wide spread and intense the complications and the sooner they appear If none have appeared within eight weeks none will appear but no case of any severity should be discharged sooner Once started complications may continue to supervene for months

Rhinorrhœa and **adenitis** have already been mentioned The latter seldom suppurates

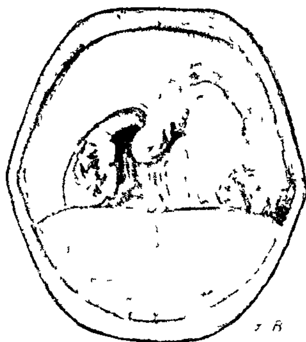


PLATE VII

Hyperto D 1 l th ı — A apı lly a l n ıng Note
tl gr œ l of tl oft] l t ɤ la n l l ft ton l
l tl sem tran pa nt prea l ng memb ne

Fa g p 136

Circulatory failure is the chief cause of death It may be peripheral or central Both commonly co exist and may appear towards the end of the first week The signs of *peripheral-failure* are those of *shock* viz an increasingly rapid pulse of falling *tension* due partly perhaps to bleeding into the capillary lake and partly to escape of fluid from the vessel walls— lymph logging' As evidence of severe vascular trauma petechiæ and ecchymoses are common in hypertoxic cases while in certain rare examples hæmorrhages from any or all of the mucous membranes may occur—*hæmorrhagic diphtheria*—an extremely fatal form

Cardiac failure is described as early when it appears within the first ten days otherwise it is termed late It seldom occurs after the sixth week (Both early and late cardiac failure may appear at appropriate times in the same case) While damage to the vagal nucleus producing tachycardia and vomiting and perhaps death cannot be excluded the chief cause of heart failure is undoubtedly the disabling impact of the toxin on the cardiac muscle fibres and the conducting bundle It is said that if 30 per cent of the former are involved failure will occur Should the bundle escape failure may occur with a normal rhythm but this is rare though bundle signs are seldom manifest until the second week Clinically peripheral and central failure merge imperceptibly into one another impossible to differentiate Commonly in the worst cases the pulse rate and blood pressure fall concurrently the rate perhaps to 40 or below and the pressure even to 65 *mm hg* At this level death may ensue if not both pulse rate and

blood pressure may rise slowly or rapidly Into this picture bundle involvement may intrude heralded usually by irregularity though it may be present only in short bursts at the outset Many courses are now possible but an increasingly rapid pulse rate with increasing irregularity is typical The heart sounds alter the first becoming as soft as or softer than the second either may be shortened or split The electrocardiograph may record anything from an inverted T wave or a simple slurring of the QRS complex to bundle branch intraventricular or complete heart block The signs change from day to day Auricular or ventricular tachycardia may appear the latter especially or the complete block is ominous It must be remembered that in diphtheria a complete block may occur with a pulse rate of 100 or over more commonly than with a bradycardia Irregularity is due mainly to extrasystoles

Clinically the failing heart is often signalized by vomiting possibly the result of the *acute cardiac dilatation* The liver is enlarged and tender Irregularity may become extreme or a sudden bradycardia ensue but nearly always a gallop rhythm coupled with extreme restlessness suppression of urine and pronounced præcordial or abdominal pain ushers in the end

Lesser degrees of cardiac involvement occur in *cases of less severity Often there may be no more* than an occasional burst of extrasystoles These serve as a useful warning since it is essential to remember that a heart which is equal to the demands of a prone patient may fail suddenly on exertion hence the importance of postural treatment The best

single guide to the state of the heart is the appearance and behaviour of the patient himself especially where the bundle has escaped and the rhythm is regular The child whose heart is affected looks limp and (apart from terminal restlessness) is quiet and subdued The child who is so lively that he cannot be kept flat almost always does well

It is well to point out that endocarditis is almost unknown in diphtheria Bruits and the like need not be looked for

Late cardiac failure technically means heart failure supervening after the tenth day but especially failure which comes on or recurs about the fifth or sixth week It is especially likely to appear in cases developing pharyngeal or respiratory paralysis It has been attributed (1) to central nervous intoxication (2) to the interference of repair tissue with the musculature and especially the conducting bundle If this last should be the true explanation it is extraordinary that permanent defects so very rarely occur More likely is explanation (3) that it is early failure incompletely recovered which has been unmasked by the strain of pharyngeal or respiratory paralysis It does not differ materially from early failure apart from being milder as a rule though in conjunction with pharyngeal or respiratory paralysis fatalities are not uncommon because of the unavoidable strain imposed on the heart muscle

Paralyses in diphtheria may follow any type of attack but are rare in purely laryngeal cases They usually occur in a definite and striking sequence While many are unquestionably the result of blood borne

toxin (e g diphtheritic pseudo tabes) others e g palatal palsy, seem to be due to direct spread to the nucleus of the nerve in question via the perineural lymphatics Thus in a case with unilateral membrane formation only that side of the palate may be subsequently paralysed Diphtheritic paralyses may be complete or incomplete and may vary in duration from a few days to several weeks When the primitive functions of swallowing and breathing are attacked there is grave danger to life

Palatal paralysis may occur from the first week onwards When late it may be the only complication The whole or only one half of the palate may be immobile As a result the nasopharynx is not shut off on swallowing and fluid regurgitates through the nose the palate does not move on saying ' ah and the explosive consonants such as p ' and b cannot be pronounced ' Billy Button ' therefore becomes Milly Mutton Palatal sensation is often lost as well

Ocular paralyses occur rather later Most frequently the ciliary muscles are involved so that the patient cannot accommodate and therefore unless myopic cannot read or see near objects clearly He may see near objects double Other third nerve palsies such as ptosis may occur Squint is often the result of sixth nerve involvement Complete ophthalmoplegia externa may occur The facial nerve may show partial or complete paralysis but it is not commonly involved

Pharyngeal paralysis is important not only of itself but because it often precedes and persists during diaphragmatic and/or intercostal paralysis It occurs

about the sixth week The earliest signs are a splut tering cough on drinking and the collection of mucus in the pharynx The superior constrictors fail to shut off the larynx during swallowing so that food or saliva may pass into it and if it is anæsthetic as it may well be in severe diphtheria or if diaphragmatic paralysis prevents coughing an inhalation pneumonia may ensue

Laryngeal paralysis sensory and motor may develop As explained it may pave the way to an inhalation pneumonia The voice becomes hoarse or may be lost

Diaphragmatic and/or intercostal paralysis often supervene on the foregoing The one or other respira tory mechanism may be singly successively or simul taneously involved If only one is affected the other overacts in compensation In diaphragmatic paralysis the patient cannot cough and respiration becomes paradoxical i e the abdomen *recedes* during inspir ation The chest wall fails to expand if the inter costals are paralysed Should both diaphragm and intercostals fail simultaneously death is inevitable in the absence of a respirator (see p 310)

Paralysis of the neck muscles is early and not un common but of the **trunk and limb** muscles is rare and late The lower limbs are most frequently involved

Sensory paralysis of the palate pharynx etc is common Loss of sensation of the glove and stocking type may also ensue

Diphtheritic pseudo tabes in which there is a loss of muscle joint and vibration sense with absent knee and ankle jerks and a tabetic gait may occur in severe

cases It may persist long after the patient has otherwise fully recovered Apart from a very exceptional hemiplegia due to thrombosis or hæmorrhage no permanent palsies ever ensue

Albuminuria is very common in the acute stages the sharper the attack the more pronounced the albuminuria Decreased secretion or **suppression** of urine is a prominent and ominous sign of circulatory failure which be it remembered may occur with kidneys that are intact Quite apart from albuminuria during the first five or six days when toxæmia is at its height albuminuria may occur about the eighth or ninth day as part of serum sickness

Diagnosis—Unless in the exceptional non respiratory forms *the swab should play a minor* part in the diagnosis of diphtheria This should be made on **clinical** grounds and where there is a doubt the diagnosis of diphtheria should be made If it proves wrong no harm is done To put it brutally (and this matter can hardly be put too forcibly) it is far easier to tell the parents of a healthy child that you sent him to hospital for observation than to explain to the parents of a corpse that you had to wait for bacteriological confirmation Yet some practitioner still has this disheartening and dismal duty to perform from time to time The refrain but the swab was negative or the result did not come back till to day accompanies almost every hypertoxic case to hospital If swabs were abolished the mortality from diphtheria would probably be halved *The first swab even in typical cases is very often negative* apart from the fact that to take a satisfactory swab single handed, from a recalcitrant child (with an

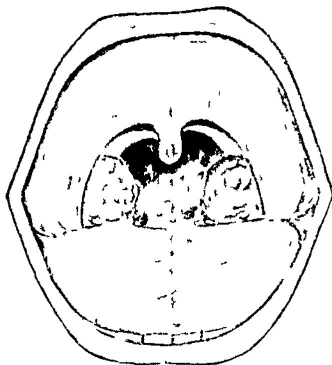


PLATE VIII

Foll[illegible] T[illegible]t

Face [illegible] Plate IX

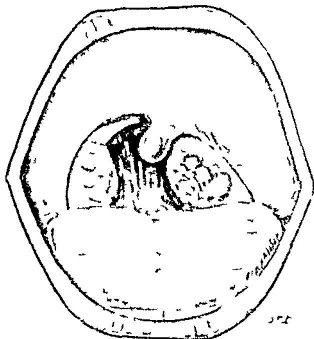


PLATE IX

Q ı y (l t t)—\ t tl e gros l w ll n l ft t ns l p ıs l n tl e la t tl ʞl t Tl re s n ıl t on tl t n l
Tl ı n y ſ t ng

F ı Fl ı (l l l l

anxious mother standing by) may be almost impossible

Diagnosis has to be made most commonly from (1) streptococcal tonsillitis (scarlatinal or otherwise) (2) quinsy (3) tonsillectomy sloughs (4) streptococcal membrane (5) ulcero necrotic conditions

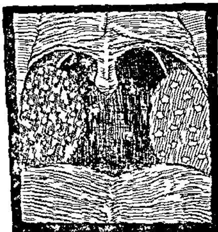


FIG 1 —Diagram to contrast follicular tonsillitis (left) with a rare form of commencing diphtheria (right) Note the injection of the tonsil in the former

including mainly Vincent s angina (6) syphilis (7) herpes (8) mumps It can often only be made on balance of probability

Points in favour of diphtheria (a) *adherent membrane* which is (b) *continuous* or in large plaques and which if detached (c) leaves a bleeding site and (d) will not break up in water (e) *extratonsillar*

spread (*f*) rapid onset (*g*) slight or no pyrexia (*h*) pallor of oropharynx and of patient—unless streptococci simultaneously active (*j*) fœtor, (*k*) copious rhinorrhœa especially if blood stained (*l*) gross œdema of palate etc (*m*) bull neck The last three may appear in intense streptococcal infections but these are rare

Points against diphtheria (*a*) mucopurulent pultaceous or granular exudate *which is* (*b*) *easily removed without bleeding and which* (*c*) *disintegrates in water* (*d*) *oropharynx generally injected* (*e*) high fever (*f*) patient flushed (*g*) much pain

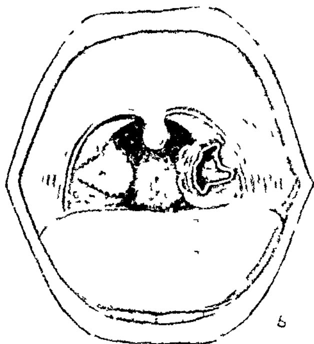
Tonsillitis if follicular is easily distinguished but not if the exudate has coalesced *The foregoing* should allow of differentiation Diphtheria may rarely begin in small patches (See Plate VIII)

Quinsy (*peritonsillar abscess*) *shows peritonsillar swelling with a little pultaceous exudate as a rule* There is trismus Fever is high the voice is typically nasal and swallowing is agonizing *It is very rare in children*, but in adults it may gave rise to confusion during the early stages (See *Plate IX*)

Tonsillectomy sloughs *unless spreading may be* ignored

Streptococcal membrane may rarely show extra tonsillar spread Unless the diagnosis is confident treat as diphtheria

Ulcero-necrotic conditions —Chief of these is **Vincent s angina,** due to the Vincent s *organisms acting* in symbiosis—*the Borrelia Vincentii* and the fusiform bacillus *A mass of heaped up glistening pultaceous* exudate much like tapioca pudding conceals a ragged and often deep ulcer on one or other tonsil



ILATI X

V i nt Ang i a (F j rocl to)—H ape l ; pultaceo
l pe t n glt t n l l l if rm el ngl t (sel e n
l c as l n n l ft t ns l

F *j j* 144

or on the faucial pillars (see Plate X) When the exudate forms a thick flat homogeneous sheet it may simulate diphtheria closely especially as it too may be afebrile It usually yields readily to an injection of N A B and the local application of Liq arsenicalis and glycerin (equal parts) or better still to the daily application of 20 per cent chromic acid Alternatively penicillin may be employed in urgent or resistant cases when it is very effective Penicillin lozenges may also be given in addition Similar ulcers may occur in the leukæmias agranulocytic angina the stomatitis due to Hg or Bi and allied conditions in most of which if not in all Vincent's organisms may be found Such ulcers may be very destructive

Syphilitic ulceration is readily detected if of the familiar snail track variety but often it is not A Wassermann should be done in every ulcerative angina

Herpes (tonsillar) may be very confusing unless herpes is present elsewhere as it usually is

Mumps shows a clean throat To diagnose mumps as diphtheria is an uncommon error but unhappily the reverse is a relatively frequent and fatal mistake

The diagnosis of laryngeal diphtheria has to be made from (1) simple catarrhal laryngitis liable to occur in many virus infections such as measles or the common cold (2) streptococcal laryngitis (often ulcerative) either primary or more often secondary to the fore going (3) foreign body in the larynx (4) laryngeal spasm whether primary as in laryngismus stridulus * or secondary to a growth or chronic ulcer (5)

* This diagnosis should be mad with considerable reserve

retropharyngeal abscess (6) œdema of the glottis and (7) pneumonia in children

In practice in children the diagnosis usually lies between *diphtheria simple catarrhal laryngitis* or *streptococcal laryngitis* If there is membrane on the tonsils diagnosis is easy otherwise it may be impossible unless one can inspect the larynx with the laryngoscope even then it may be difficult Unless there are Kopliks present a croup' should always be given diphtheria antitoxin The history is often useful some children develop laryngitis every time they catch cold *Laryngismus stridulus* is a sudden acute spasm in a previously (and subsequently) normal child under two years *Growths* chronic *ulcers* and *œdema of the glottis* are usually diagnosable from the history and often show an associated condition excepting papilloma they are rare in children *Retropharyngeal abscess* can be seen or felt it causes *a stertorous type of breathing rather than stridor* Pneumonia in children sometimes causes stridor which however *is expiratory and not inspiratory* A foreign body should give a characteristic history The student should make every effort to hear a genuine croupy cough and stridor once heard they are not easily forgotten The stertor like breathing of pharyngeal obstruction is the most frequent cause of alarm and confusion

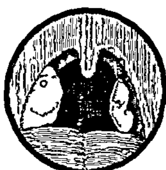
Prognosis —In all forms of diphtheria prognosis depends directly on the promptness with which serum is administered Most deaths occur in cases that do not come under treatment until the fourth or fifth day or later **After the fifth day antitoxin is largely ineffective** Age is also a factor—the younger the patient the worse

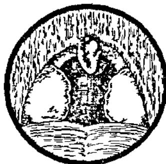
the outlook So too is nutrition—undernourished children fare badly In any given case *signs of good import* are (1) good colour and pulse (2) child alert and lively (3) little or no albuminuria (4) complications late in onset (5) good sedimentation rate *Signs of bad import* (1) pallor (2) hæmorrhage (3) vomiting (4) tachycardia (5) bradycardia (6) much albuminuria (7) anuria enlarged liver and gallop rhythm—these portend a fatality

One point which should be remembered is that no matter how desperate the patient s condition or how severe or widespread the complications if the patient recovers no permanent damage ever remains unless in the most rare and exceptional cases This should always be explained to the parents or relatives to set their minds at rest on that score at any rate

Treatment—Serum is the first essential—intramuscularly or intravenously—at the earliest possible moment This arrests spread of membrane and neutralizes circulating toxin preventing the fixation of any more in the tissues Dosage varies (See Fig 13) The smallest dose should be 8000 units for a chronic nose and 16 000 for a mild first day faucial Each day thereafter dosage should be increased subject to a limit in all of 100 000 Note that children require more not less antitoxin than adults The whole amount should be given at once and in severe cases part at least intravenously with 25 per cent glucose in saline In purely laryngeal cases 3 000 units is ample If prostration is severe glucose with insulin may be required but the blood sugar must be checked by frequent estimations Glucose by mouth may be given for a fortnight







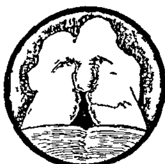


FIG 13 —Do age of ant toxin 1 pend primar ly on the degree of toxæmia Add tional factors are (1) the extent of membrane (2) the day of d seas and (3) presence or abs nce of rhinorrhœa or laryngeal involvement The above are adequate O er 50 000 un ts part sl ould be gi en intravenously (see p 147)

What is the role of penicillin in diphtheria? So far it has not been satisfactorily established. Since the C. diphtheriæ as well as the streptococcus is penicillin sensitive *prima facie* there would seem to be good grounds for using it in all sharp cases both systemically and locally. But while penicillin can destroy the organism it cannot neutralize toxin so that it cannot be expected to *replace* antitoxin. At present therefore all we can say is that the wisest course is to employ both in all really sharp cases.

The same applies to a lesser extent to the sulphonamides. Wherever there is evidence of streptococcal proliferation—e.g. high fever, streptococcal tongue, etc.—if penicillin is not available the sulphonamides should be employed. If penicillin is to be had then of course the sulphonamides would be unnecessary.

Postural* treatment is the second essential except in chronic anterior nasal cases. The heart must be spared. The patient is put to bed flat without pillows, and if there is any suggestion of heart failure the foot of the bed must be raised to ensure the blood supply to the vital centres in the mid brain. The patient must do nothing whatever for himself and enemas are employed to save him straining at stool. He may have to be kept flat for one, two, three or even six weeks depending on the severity of the case, after which one, two and three pillows are allowed at weekly or fortnightly intervals. He sits up in a chair for increasing periods before being allowed to walk. A practical point of some importance at this stage is that on polished wooden floors rubber shoes are

* The mother of a suspected case should be told to put him to bed at once. She should never be told to bring him to hospital again."

advisable to avoid accidents since he is often very ataxic

While membrane persists discharges are swabbed clean and the toilet of the mouth is performed but local treatment is otherwise contra indicated and no attempt should ever be made to pull membrane away The diet is fluid in the acute stages thereafter it may be mixed provided that feeds are small and do not embarrass the heart which must be most carefully watched for extrasystoles, tachycardia or bradycardia The child s appearance and behaviour must be carefully noted At the first hint of cardiac involvement he must be placed flat *Exercise tolerance tests are dangerous a good test is to instruct him to take a deep breath If normal sinus arrythmia occurs the bundle is probably normal If the rate is unaffected, suspect it This simple procedure may also provoke* extrasystoles and thus unmask a latent lesion

Treatment of Complications—In the initial collapse the patient must be kept warm if necessary by the electric cradle but care should be taken to see that he is not overheated As adrenal insufficiency may be a factor eucortone or adrenalin may be administered In established *heart failure* there is little or nothing that we can do though stimulants such as coramine adrenalin camphor in oil brandy etc may be of temporary value Hot packs to the praecordium and even morphia may be required to relieve the terminal pain

The paralyses in general neither demand nor are they susceptible of treatment as a rule *Palatal palsy* may require thickened fluids *Pharyngeal* and *laryngeal* paralyses are dangerous because of the risk of

inhalation pneumonia Both will demand nasal feeding The foot of the bed must be raised and the patient laid on his side to allow the mucus and saliva to drain away through the nostrils Suction by means of a catheter through the nose into the nasopharynx may be of value Atropin may be employed to check the secretions but is highly unpleasant

Respiratory palsies often supervene on the former Since the foot of the bed must be kept elevated the weight of the viscera may embarrass the diaphragm If the palsy is severe death may occur in the absence of a respirator (see p 310) As transfer to a respirator may be frightening to some children it is a good plan to persuade a convalescent volunteer to go into the respirator first in order to inspire the patient with confidence Resistance or struggling on the part of such a gravely ill child should at all costs be avoided

Treatment of Laryngeal Diphtheria

In addition to serum and postural treatment the obstruction must be dealt with Steam with or without a tent and fomentations to the larynx are usually employed If available the administration of helium 75 per cent with oxygen 25 per cent through a B L B mask may allow operation to be avoided by minimizing the effects of the obstruction Most careful watch must be kept on the pulse Should this increase in rate and lose its tension and especially if it should intermit measures of relief must be considered Particularly if the child begins to throw himself about *Never wait until pallid cyanosis appears* Three courses are open (*a*) to

perform suction (*b*) to intubate or (*c*) to perform tracheotomy

Suction —This requires a suction motor coupled through a bottle containing water to a rubber tube carrying the metal 'sucker' The child is placed *on the operating table with his head overhanging* the edge The nurse supports it in semi extension A gag or bite is placed in the mouth the direct laryngoscope is introduced the epiglottis hooked forward and the larynx inspected The motor is now switched on and the metal tube is introduced through the laryngoscope and through the glottis as the child inspires Suction is now applied The sucker is rapidly withdrawn from the glottis and re introduced several times The tube is then withdrawn and placed in a bowl of water which on being aspirated *will clear the suction tubing so that the amount and* character of the debris from the glottis can be ex amined At the same time the degree of relief afforded the patient can be assessed

Intubation —This may be done through the laryngo scope by means of a special long forceps (*direct*) or *indirectly* by the sense of touch The child is placed low down on a bed or cot with the head extended and restrained (*The tracheotomy instruments are to hand*) A gag is introduced The operator then passes his left index finger into the child s pharynx hooks forward the epiglottis and feels the arytenoids With the other hand he passes the well lubricated tube on the introducer down in front of the finger and manœuvres it into a direct line with the larynx Then *as the child inspires he releases the catch and* slips the tube into the larynx tapping it gently home

with the left forefinger The operation requires much practice not easy to obtain No more than three attempts in all should be made as the process is exhausting to the child If by that time no success has been achieved tracheotomy should be performed If the intubation tube carries—as it should—a string this may be brought out and fastened to the cheek with strapping

Extubation —The operator places his left thumb in front of the trachea just below the larynx and his right hand on the occiput The head should be fully extended with the mouth open Suddenly he presses his thumb backwards simultaneously smartly flexing the head with the result that the tube is forced into the mouth Removal of the tube with a special extubator may be very difficult but is very easy by the string (if one is present)

Aftercare —Return of obstruction must be watched for The tube may be coughed out or coughed up and swallowed If either occurs reintubation must be performed if necessary If it recurs frequently tracheotomy may have to be done Otherwise the tube is removed in two or three days Often when the œdema subsides the child may cough the tube out and it may be found that it is not necessary to put it back

Tracheotomy —The child is pinned in a blanket and laid on the table with a sandbag so adjusted beneath his shoulders as to give the maximum extension of the head and throw the trachea well forward If there is time an anæsthetic may be employed—either local or general Local anæsthesia however obliterates the landmarks which is a very

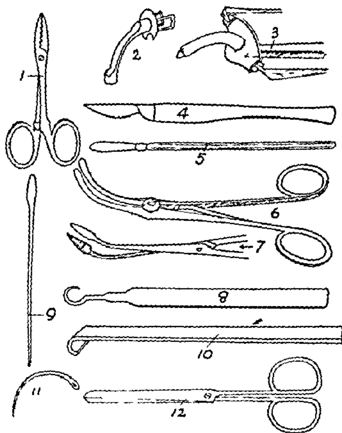


Fig 14—Instruments for tracheotomy Note (2) inner tube (3) outer tube on pilot (6) dilators (7) membrane forceps (8) tracheal hook and (10) retractor

serious matter in small babies As for general anaesthesia inhalational anaesthetics require great skill in administration while one s own experience suggests that such children tolerate intravenous anaesthetics badly In any case tracheotomy is usually an emergency operation and indeed often one has to operate on a child that is to all appearances moribund Speed is the essence of the contract One assistant stands to his left steadying the pelvis and controlling the arms another holds the head firmly and steadily in the midline This is essential if the head deviates the trachea may be entered at the side Should this happen in a small baby it may not be possible to get a tube in at all because the trachea may be no bigger than a pencil In adults a choice of high or low tracheotomy may be made in children this does not exist Again the trachea in adults is rigid and fixed in babies it is collapsible and moves about quite freely The operator steadies the larynx with the thumb and *second* finger of his left hand and places the index finger on the cricoid He then makes his skin incision starting just below his index finger and therefore below the cricoid An assistant swabs with adrenalin and hands the instruments If the isthmus of the thyroid obstructs it will have to be pushed up or down out of the way with the knife handle The trachea *must* be felt with the left forefinger before it is opened preferably it should be seen as well The trachea is divided through the first two rings *The knife is kept in the trachea* * and rotated to point

* This method has its defects—and its critics—but if carried out smoothly and quickly gives in my own experience much the best result

laterally The left hand is only then removed to pass the dilators down along the knife blade Only when these are *in situ* and opened is the knife removed The *child usually begins a paroxysm of coughing as soon* as the trachea is opened and he should be allowed to quieten down before the tube is inserted The pilot carrying the tube is held horizontally *at right angles* to the skin incision the tip is passed down between *the dilator blades into the trachea the dilators being*

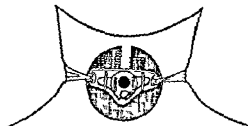


Fig 17 —Tracheotomy tube in situ

simultaneously withdrawn The pilot handle is then rotated towards the midline and elevated to the vertical at the same time with the right hand the tube steadied with the left and the pilot withdrawn The tube is tied *in situ* and the inner tube inserted

Common Causes of Failure —(*a*) Head not held firmly in midline (*b*) trachea not steadied and slips away both (*a*) and (*b*) lead to incisions into the trachea that are not in the midline (*c*) losing the opening—knife removed before dilators are in position —a most harassing mistake that can very easily be made (*d*) *attempting to insert pilot with handle in line with* trachea instead of *at right angles* (*e*) rarely—a

too forcible incision which passes through the trachea into the œsophagus (The trachea in children cuts very easily) Dilators too widely opened may paradoxically block the trachea

The operation must be done rapidly unless an anæsthetic is used Hæmorrhage is often profuse it usually ceases as soon as an airway is established But if there is much delay blood in quantity may get into the trachea with fatal results Holding the child head downwards may sometimes drain it away

Aftercare —The child is put back to a warmed bed and usually falls asleep at once If he is shocked brandy may be given He should be *left alone* and not meddled with unnecessarily Symptoms of obstruction may recur due to a displaced or dislocated tube or to blockage If they are not relieved by replacing or cleaning the inner tube the obstruction must be low down The outer tube should be removed the dilators inserted and attempts made to remove the obstruction with membrane forceps or suction may be applied through a catheter which can be passed down even into the right bronchus This is often very successful Cases which cough up abundant mucus (and membrane) do well dry cases do badly Often in these dry cases there is a bronchopneumonia diphtheritic or streptococcal Spraying the trachea with weak sterile soda bic or instilling olive oil may help but such cases offer a gloomy prognosis They are rarer since the antibiotics have appeared

The tube should be removed normally in about forty-eight hours if during sleep so much the better since much subsequent distress often manifested on removal is psychological A good nurse who can

allay the patient's fears is invaluable If distress is well founded removal may be postponed for a further twenty four to forty-eight hours after which a determined effort must be made otherwise it may prove very difficult to get the tube out at all If there is still genuine distress without it it is probable that the cricoid cartilage or even it may be the thyroid cartilage has been cut at operation producing laryngeal stenosis If so the tube may have to stay in permanently

Evaluation of Various Methods —Briefly it may be said that tracheotomy quite definitely relieves laryngeal and upper tracheal obstruction Skilfully performed it should leave no permanent ill effects and if the instruments are available it may be done anywhere On the other hand it is a difficult and dangerous operation even in the hands of an expert and is inevitably attended with much shock which alone may prove fatal Intubation causes less shock and is not dangerous but requires much skill and practice If the obstruction is subglottic it may not relieve it But the great objection to intubation is that the child may cough the tube out and if a skilled intubator is not at hand to replace it may die It is therefore practicable only in hospitals where skilled assistance is at hand and is useless in private practice

Of suction it may be said that in certain types of cases it is very successful and it is not dangerous to life *though it must be highly unpleasant* Further intubation via the laryngoscope is easy But as one suction rarely gives permanent relief it has to be repeated and it may prove very exhausting Where it fails as with intubation tracheotomy must be

resorted to which tends to be so delayed that the child fails to withstand the operation

In skilled hands (and this includes not merely the operator but the nursing staff) any method can be made to give excellent results but fortunately or unfortunately nowadays experience is hard to come by Fewer and fewer cases of laryngeal obstruction due to diphtheria come to operation year by year thanks mainly to immunisation For general purposes tracheotomy is usually regarded as the method of choice in this country though alternative methods have ardent supporters

Prevention of Diphtheria

Diphtheria is now a preventable disease In certain cities of North America it has been practically abolished The procedure is neither difficult nor dangerous

The Schick test roughly determines susceptibility to diphtheria 0 2 c c of a dilution of toxin (equivalent to $\frac{1}{50}$ of the *minimum lethal dose**) is injected intradermally into the left forearm a similar amount of heat inactivated toxin being injected into the right The result is read one three and seven days later Four results are possible (1) a zone of erythema the size of sixpence or a shilling occurs and persists on the test (left) arm but none on the control arm This result is *positive* and indicates susceptibility (2) there is no result on either arm—this is *negative*

* The M L D of toxin is the amount which will cause the death of a 250 gramme guinea pig in precisely four days.

(3) both arms show a reaction that on test arm being larger and more persistent Part of the reaction on the test arm and all that on the control is due to proteins (other than the toxin) present in the solution and is called a *pseudo* reaction The result is therefore *pseudo* and *positive* (4) both arms show a transient reaction of the same size This result is *pseudo* and *negative* (3) is therefore positive and (4) negative Positive results may show for a month or more They imply that the person has less than $\frac{1}{30}$ of a unit of antitoxin in his blood which is believed to be the minimum for protection

Immunization — Diphtheria Prophylactic' is the filtrate of a broth culture which has been treated in various ways As a preliminary formalin is added and the mixture incubated This greatly reduces its toxicity but leaves its antigenic power relatively unimpaired It it then known as (1) **F T** (Formol Toxoid) This is a good immunizing agent though liable to produce reactions in children over eight years because of the bacterial protein it contains Two intramuscular injections are usual in Canada where it is preferred (2) **T A M** This is F T to which antitoxin is added as a precaution But it may produce similar reactions to F T with the grave additional disadvantage of rendering the recipient serum sensitive (unless the antitoxin has been prepared with goat serum) Three injections are required as of (3) **T A F** where antitoxin is mixed with F T to the point of flocculation Much harmful protein is eliminated in preparation and it is safer than T A M but more expensive It is similarly serum sensitizing (4) **A P T** is F T to which alum has been added forming aluminium hydroxide

to which the toxin is adsorbed The precipitate is well washed so that much protein is removed It is not of course serum sensitizing It is a potent antigen so much so that one large or two small injections often suffice The one shot method is not to be recommended however If combined with subsequent weekly in intranasal installations of F F it is said to give 99 per cent immunes Protamine toxoid which has been recently introduced is said to be better than A P 1

Immunisation Procedure —Do a preliminary Schick test Read in one week If positive give A P T 0 5 c c at once and follow with 0 5 c.c in four weeks Or give three doses of T A F 1 c c at three week intervals

Whatever method be employed a Schick test should be performed about twelve weeks later If still positive the course should be repeated—with caution if T A M or T A F has been used A P T is probably the best of all Immunity so produced should last for some years and even if subsequently diphtheria be contracted however extensive the membrane there is a marked absence of toxæmia—and therefore of complications

Nowadays the general procedure is to carry out immunisation at about the age of twelve months and some four years later to give a boosting dose before the child commences school where the risks of infection will manifestly be greatly increased

The Moloney test may be done in the same way as a Schick but with a dilute solution of the immunizing agent it is intended to use instead of dilute diphtheria toxin It s purpose is to find out if the patient is likely to react to the agent proposed An area of erythema—like a pseudo Schick reaction—will occur

within twenty four to forty eight hours if the test is positive in which case some other antigen must be employed

Control of Outbreaks.—Look for the source—often a case of chronic anterior nasal diphtheria isolate and treat if found Take swabs and do Schick tests all round Four classes will result (1) Schick negative culture negative—do nothing (2) Schick negative and culture positive—segregate, investigate virulence of organisms (3) Schick positive culture positive—segregate and give small dose of antitoxin as he may be incubating the disease (4) Schick positive culture negative—watch closely If any suspicion of clinical signs in either (3) or (4) give antitoxin in quantity at once If signs in (1) or (2) send to hospital for investigation

SECTION VII

THE INTESTINAL (INGESTION) INFECTIONS

ENTERIC

ENTERIC or typhoid fever is an acute specific infectious disease characterized by continued fever a rash and ulceration of the intestine

Cause—The *Eberthella typhi* and the allied organisms—*E paratyphosi* A B and C *E typhi* and *Para B* are commonest in this country All four organisms cause diseases so like one another as to be clinically indistinguishable (as a rule) which are known collectively as the *enterica* Of the E typhi alone many different types have been distinguished by bacteriophage comparable to the types of streptococci established by agglutination

Incidence.—Apart from localized epidemics the incidence is steadily declining It is commonest between the ages of ten and twenty years while under five and over fifty it is rare

Mode of Spread—Infection occurs by swallowing food or drink contaminated by the bacilli As the dejecta of sufferers from the disease contain the organisms and as some who recover remain infectious for life infection may first of all arise from contact with the excreta or fomites of a patient or carrier Secondly if the dejecta are improperly disposed of or if the drains are defective organisms may infiltrate a well or other source of water supply Thirdly if sewage

is insufficiently treated it may contaminate a river or the sea nearby so that infected oysters, and such vegetables as cress may act as agents of spread Fourthly the fly settling on excreta and then on food may propagate infection Perhaps most dangerous of all is the carrier (often as the result of an unrecognized ambulant attack) employed in a food shop or on a farm or in a dairy since the organisms may multiply readily in milk without altering the taste Milk out breaks are often most explosive " in character In brief fingers flies food and fomites are the channels of transmission the commonest foods " being milk water shell fish and vegetables But bread handled by a carrier or even cheese made by a carrier, can transmit the disease while a carrier acting as cook (like the notorious Typhoid Mary) can wreak havoc wherever she goes Remember that freezing is no protection—ice or ice cream may initiate an epidemic

Pathology—The organisms have a predilection for the Peyer s patches of the small intestine whence they pass perhaps via the mesenteric glands the spleen and ultimately the thoracic duct to the blood These patches the lymph nodes generally, and the bone marrow become crowded with large mono nuclear phagocytes and small lymphocytes to the exclusion of the polymorphonuclears and eosinophils This is reflected in the blood where there is a leuco penia affecting especially the latter cells (unless extraperitoneal suppuration occurs) The Peyer s patches become swollen and ultimately necrotic These necrotic sloughs next separate exposing an ulcer which may erode the bowel walls even to the

peritoneum As this process comes to a head towards the end of the second week and as the lymphoid patches are very vascular perforation or hæmorrhage may be apprehended from that time onward These accidents or the fear of them dominate the mind of the clinician the more so since they do not at all correspond with the severity of the case Death from toxæmia may ensue with trivial intestinal lesions and death from hæmorrhage or perforation can occur in a case so little toxic as to be ambulant

Morbid Anatomy —The ulcers are oval with the long axis in the long axis of the bowel Normally they heal to leave no scar The *spleen* is enlarged and crowded with erythrocytes The *kidney* may show clumps of bacilli and the *liver* a focal necrosis The *gall bladder* is infected early and since unlike most organisms the typhoid group can multiply in bile it offers a congenial environment and often becomes a more or less permanent reservoir of infection The *lungs* often show a bronchopneumonia The *veins* are often thrombosed The *tibia ribs sternum* or *vertebræ* may be attacked resulting in periostitis or suppuration—often occurring long after the original attack The *rose spots* are unusual in that they are bacterial emboli in the skin vessels from which the organisms may be recovered

Incubation Period —Ten to fourteen days

Segregation Period —Until three consecutive specimens of stools *and urine* are negative

Course —Enteric is the classic example of a fever exhibiting normally the incubation period the stage of invasion the stage of advance and the stage of decline but in the sporadic cases seen in this country

to-day, classical examples are relatively rare. In particular the rash is often absent (though Para B forms occasionally present a profuse eruption) and the pyrexia often follows a deceptive course. In obscure pyrexia of any duration enteric should be one of the first suspicions.

Stage of Invasion.—Following the incubation period *the stage of invasion sets in, often insidious and undramatic.* The patient merely feels "off colour" and apathetic. He may have a persistent headache and a poor appetite, but as often as not complains of nothing abdominal. If he does, it is usually only of vague discomfort. Rarely there may be diarrhœa; more often he is constipated. His tongue is furred and he may feel chilly, though an actual rigor is rare. *These symptoms persist and increase in severity so as finally to drive him to bed. It will now be found that though there is pyrexia there is no corresponding tachycardia.* The temperature mounts classically in "staircase" fashion—i.e. a rise of a degree or more in the evening, followed by a fall of half a degree next morning—until it reaches a maximum of 102° F. or *thereabouts, where, with slight diurnal variations, it* remains. In sporadic cases the physician may be puzzled, even if he has detected the enlarged spleen, since there are no other signs; but note that at this stage a positive blood culture can be readily obtained. Further, the pulse is usually dicrotic—a curious but characteristic and fairly constant sign.

Stage of Advance.—This corresponds in classical examples with the second week of the disease. The pyrexia is high, remitting but little, the pulse dicrotic and relatively slow, and the "clouding" of the mind

—suggested by the nomenclature of the disease—is well in evidence The lips are coated with sordes the tongue is brown dry and fissured the abdomen is tumid and the spleen palpable About the seventh day the rash appears Typically it consists of sparse widely separate rose pink maculopapules on the lower chest abdomen and back which disappear on pressure Often the rash is absent Rarely it may be profuse on the body generally though it is seldom seen

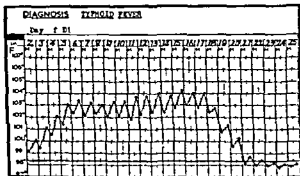


Fig 16—Enteric

on the face It appears in crops which commonly overlap each lesion persisting for three or four days (While a most useful diagnostic sign the rash has little prognostic significance An absent rash does not imply a mild case) Finally there are the characteristic stools which when typical are foul frequent bulky and pea soup in colour and consistency

The close of the second week and the beginning of the third may be regarded as the acme of the disease The sloughs in the bowel are beginning to separate

and it is now that the dreaded complications—hæmorrhage and perforation—may be apprehended But quite apart from such accidents the toxæmia is at its height and in cases fatal from that cause the patient sinks into the "typhoid state"—a condition of profound intoxication He usually lies on his back, too weak to turn on his side His eyes may be half closed or open and wandering but the dilated pupils show that he is not accommodating or indeed seeing anything consciously In his emaciated exhausted face the eyes often stand out abnormally large and alive He may be quiet but more often he is continually whispering and muttering to himself picking aimlessly at the bedclothes with wavering trembling hands These purposeless finger movements are known as *carphology* the tremors as *subsultus tendinum*, and the whole condition as *coma vigil* This last is an apt term since for all his activities the patient is quite unconscious Soon the movements weaken and cease the coma deepens and death ensues

Stage of Decline—In more favourable cases about the middle of the third week the temperature begins to remit with a larger fall than subsequent rise and a gradual termination of the fever by a slow lysis ensues By about the end of the third week the patient should be afebrile He has lost considerable weight is seriously enfeebled and ravenously hungry but his mind at last is clear Convalescence now begins it is likely to be prolonged Apart from sequelæ elderly patients may never recover their former vigour

Relapse—This is an extraordinary feature of enteric Within a fortnight or so of defervescence, in

roughly one case in ten the disease sets in anew following usually a milder though occasionally a more severe course than the original attack This is described as a recurrent relapse In rare cases a *concurrent* relapse appears before the initial fever has wholly disappeared Since second attacks of enteric are otherwise exceedingly rare the mechanism of these relapses is inexplicable unless a reinfection with an organism of a different type has occurred analogous to those encountered in scarlet fever or the antigens of the bacillus have undergone some mysterious metamorphosis (Recent work with bacteriophage as noted above shows that in fact several different types of the B typhosum exist) In most cases be it noted relapse occurs when the defence mechanism is at the peak of efficiency i e when the patient has repelled the attack and has reached the convalescent stage Obviously in the light of more recent knowledge of antigenic constitution further study of relapses is imperative

Varieties — While the foregoing is an account of the disease as classically described many deviations from this pattern will be encountered Further from the point of view of the essential time relations it is often exceedingly difficult to decide exactly when the attack began within a limit of several days Very many cases seem to overrun the three weeks giving rise to a prolonged form others again are curtailed Four types are usually distinguished — (*a*) the *ambulant* type in which the patient does not at first feel at all ill (though he may later suffer perforation) (*b*) an *abortive* type (*c*) the *classical* type just described and (*d*) a *hypertoxic* type in which

prostration is extreme from the outset and death supervenes early These are merely ascending grades of severity Cases also occur in which the diagnosis is masked by some coincident manifestation e g pneumonia nephritis meningitis such cases may be referred to as the *pneumonic* '*nephritic* or *meningeal* type A very rare *hæmorrhagic* type has also been described

Diagnosis —This is not difficult provided only that enteric be suspected Classical cases of sustained pyrexia showing a staircase rise which manifest an enlarged spleen and later an appropriate rash and pea soup stools are rare The clinician will have to be prepared to detect cases which develop no rash or merely an equivocal one and which moreover may show *constipation* throughout The sustained pyrexia and enlarged spleen are however constant in all but the very mildest cases On the other hand symptoms may occasionally suggest a diagnosis of pneumonia meningitis or the acute abdomen Generally however miliary tuberculosis enclosed sepsis malignant endocarditis or undulant fever most commonly confuse and the differentiation may clinically be extremely difficult Fortunately the laboratory comes to our aid in no uncertain fashion Five investigations can be made —(1) blood count (2) blood culture (3) agglutination reactions (4) isolation of the organisms from the stools and urine and (5) the diazo reaction

(1) **Blood Count** —This shows a leucopenia especially affecting the polynuclears throughout the disease unless in the very late stages when extra abdominal suppuration has occurred e g bone abscess Hence

septic infections can be excluded by blood count in the early stages

(2) **Blood Culture**.—This is positive (and diagnostic) in 90 per cent of cases for the first seven to ten days and in 50 per cent of cases for the next week. About 5 c.c. of blood should be added to a bottle containing bile broth and sent to the laboratory. At a later stage sternal puncture may give a positive culture.

(3) **Agglutination Reactions**.—As noted there are two classes of agglutinins developed to the antigens contained in typhoid organisms—(1) the flagellar and (2) the somatic. The flagellar comprise (*a*) a type agglutinin which will react with other organisms in the group and (*b*) an agglutinin specific to the organism. Either may predominate. Both varieties of flagellar agglutinins give a coarse flaking deposit in the tube; they are prone to linger in the blood for years following prophylactic typhoid vaccination and furthermore in febrile states to increase in titre—the *anamnestic* reaction. Therefore they are not to be accepted in diagnosis even if they show a rising titre during an illness. On the other hand the somatic O and especially the somatic Vi agglutinins represent (according to Felix) the body's response to the genuine toxic and invasive antigens of the bacillus; such agglutinins if present in a dilution of $\frac{1}{60}$ or over are diagnostic (unless the patient has been inoculated within the preceding two months) and if present *in rising titre* are conclusive. A few c.c. of blood should therefore be withheld from the culture specimen and forwarded for a determination of the O and Vi titres. These may be very low during the first week but it is essential to have a record of

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them for future reference After the tenth day the titre mounts reaching its maximum about the twenty first day It then falls unless in carriers when it persists In general the "O" and "Vi" give the diagnosis (of enteric) and the "H" specific agglutinins give the differential diagnosis In some cases agglutinins are unaccountably absent—perhaps in those in which relapse may occur

Isolation of the organisms from the stools by plating out on McConkey or similar medium can be carried out from the second week onward or earlier on the brilliant green eosin agar medium They are often profuse following a purge and probably derive not from the ulcers but from the gall bladder They may also be isolated from the urine and persist in both excretions for many weeks In carriers the stools may be infectious for life

Diazo Reaction —If an equal quantity of diazo reagent and urine from a patient in the second week of enteric be mixed and rendered strongly alkaline with ammonia a ruby red colour will be produced This reaction is present in many febrile conditions and though always described is little used

To Sum Up —During the first ten days blood culture and stool culture on special media thereafter the Widal and culture from the stools on McConkey A blood count at any time is helpful occasionally the diazo reaction may be of service

Prognosis —This is the better the younger the patient In the previously inoculated it is also improved It is said to be better in *para* as against *typhosus* infections but this is not unanimously accepted In any given case the degree of toxæmia is

the decisive factor though this does not influence the probability of perforation or hæmorrhage All cases of enteric however mild are potentially dangerous

Enteric in Children —In children under five years enteric may assume a very atypical form Thus it may appear as an acute gastro-enteritis or a septic æmia or signs of bronchopneumonia or of meningitis may obscure the picture This last may be merely a meningism or a true meningitis due to enteric organisms The rash too may be sparse—or most profuse

Complications

These may be legion and many are of little practical significance There are two however of outstanding importance which directly threaten life both of which arise from the same source namely the separation of the sloughs While this may begin early in the second week it is towards the end of the second and during the third week that most serious accidents occur

Hæmorrhage —Small amounts of blood may escape into the intestine in the early stages these need cause no anxiety Severe hæmorrhage usually due to the erosion of a sizeable blood vessel occurs as stated above about the end of the third week though it may occur earlier and is a most insidious complication since the first notice we may have of it is a motion consisting almost entirely of pure blood In the worst cases signs of shock may rapidly develop for no visible reason—i e the patient is pale restless and collapsed the pulse is rapid and small and

air hunger is pronounced The temperature may also fall, though not invariably Whenever hæmorrhage occurs the outlook is grave One large hæmorrhage may be survived but repeated ones are usually fatal

Perforation occurs during the third week as a rule the separating slough carrying with it a portion of the peritoneal coat This results in the escape of bowel contents into the general peritoneal cavity and therefore in general peritonitis The onset of a sudden severe pain in the abdomen is usually the first sign the site of the pain varying with that of the perforation though the student is warned that pain may sometimes be absent Very shortly the signs of general peritonitis supervene—tenderness and rigidity of the overlying muscles often distension and perhaps obliteration of liver dullness A rigor may occur The patient may lie with knees drawn up a grey pallor pervades the face and the expression becomes increasingly more anxious Vomiting may be most distressing If collapsed the additional signs of that condition will appear Rarely the signs will remit only to recur later with enhanced intensity

As a rule the pain is in the region of the right iliac fossa since most perforations occur in the last few feet of the small bowel but in rare cases the site of perforation may lie in the ascending colon or even in the appendix itself In children perforation is almost unknown

Meteorism may precede or follow perforation it is a grave complication because of the resultant cardiac embarrassment and is of evil omen

Peritonitis without perforation may also occur by direct spread across the peritoneal coat of the bowel

or from suppuration of a mesenteric gland **Pneumonia**—lobar or lobular—is not rare and **hypostatic** pneumonia is to be feared in the severe cases *Laryngeal ulceration* is a common complication in some parts of the world Transient but temporarily complete **deafness** may be met with **Nephritis** is rare so too is a clear cut **carditis** **Cholecystitis** in some degree is probably invariable as the organisms find the gall bladder a most congenial habitat so congenial indeed that they may persist there for life Suppurative cholecystitis is rare but chronic cholecystitis accompanying subsequent cholelithiasis is very common in after years **Venous thrombosis** usually involving the femoral or popliteal vein is a complication of convalescence So too is **periostitis** or **suppurative osteitis** of the tibia ribs sternum or vertebræ (typhoid spine) these conditions may suddenly flare up months or years later when living virulent bacilli may be isolated from the lesions They are accompanied by a polynucleosis **Parotid abscess** arising from a neglected mouth is nowadays rare

Treatment and Management—Apart from Felix s serum which has not fulfilled expectations though useful to reduce toxæmia we are quite without a specific agent in the treatment of enteric Neither penicillin alone nor the sulphonamides alone have appeared to have any influence on the disease Very good results however have been reported from *penicillin coupled with the sulphonamides and* it seems likely that one or other of the new antibiotics will provide us with an effective weapon (Streptomycin promises very well) Until that happens we are driven back on the classical management of the

febrile state Chief amongst the measures advocated is *hydrotherapy* This should take the form of sponging or of immersion in a bath The latter is only really practicable in hospital Neither cold baths nor cold sponging have ever found much favour in this country, but the tepid bath has many advocates Tepid *sponging* is however much more widely practised It should be carried out daily or twice daily as a routine and four hourly where the degree of toxæmia warrants it At the same time it must be remembered that it has no specific curative action and its application should therefore be elastic and not rigid It would be folly for instance to wake a sleeping patient merely to sponge him

The liability to hæmorrhage or perforation especially at the danger period should always be borne in mind The patient should be kept quiet and any vigorous movement in bed should be discouraged On no account should he be permitted to get up or sit up however mild the case The skin and pressure points will need special attention and the toilet of the mouth should be scrupulously carried out

The diet in enteric is still much in dispute That masochistic attitude in medicine which is as old as the race and is based on the irrational belief that anything beneficial must of necessity be unpleasant and painful dies hard in most of us For many years the febrile patient was not even given enough water and for many years more the patient with enteric was allowed nothing but milk presumably for fear that anything more solid might induce perforation with the result that if and when he survived to convalescence he was an emaciated wreck Now,

while frankly indigestible commodities such as raw or undercooked vegetables can be of little benefit and such penetrating articles as fruit pips might actually be dangerous it is obvious that the vast majority of foodstuffs are reduced to a fluid or semi fluid state long before they traverse the ulcerated area It is especially difficult to see why meat even if minced is still taboo Certain writers it is true maintain that the surest way to provoke perforation and hæmorrhage is to starve the patient and feed him generously but this attitude is far from general There can be no question that the high protein wastage inseparable from prolonged fever demands replace ment if the patient s resistance is to be maintained to say nothing of the vitamin intake If we are to restrict the diet let us do so because of the fever and resultant gastritis and not because there are ulcers six hours or more distant in time from the stomach contents In the early stages milk abundance of water with glucose bread and butter mashed potato eggs (raw poached or lightly boiled) jellies custards and so on may be allowed depending on the patient s appetite tolerance and idiosyncrasies Should he demand still more well filleted fish minced chicken and the like can be given and if necessary red meat—provided he can digest it This applies to the milder and less toxic cases obviously a hypertoxic patient who may be delirious is not likely to take much of anything even fluids But each case must be judged on its merits That starvation or the interdiction of this or that article of diet is an essential in the treatment of enteric is an idea it were well to abandon

The regulation of the bowels also demands consideration The student must remember that the intestinal tube is dangerously thin at many points None the less it must continue to function Anything which greatly increases the force of peristalsis might well determine a doubtful perforation Strong purgatives should be taboo and indeed purgatives of any kind are best avoided A carefully given enema is more desirable and at least as efficacious

Medicinally there is little to give beyond drugs to relieve symptoms as they arise Pain or headache may require aspirin, and insomnia should be countered promptly and decisively But drugs on the whole are of minor importance In this more than any disease the physician must rely on the nurse She must prevent any exertion or violent movement change the patient s posture should he lie too constantly on his back (lest hypostatic pneumonia develop) safeguard the skin and anticipate and prevent any straining at stool Neglect of any of these measures may have serious results

To prevent spread of infection the strictest aseptic nursing must be carried out The frequent use of the wash basin is the best preventive The stools should be covered with disinfectant and allowed to stand suitably covered before emptying into the closet and the bed pan or urine bottle should then be sterilized The attendants ought to have been inoculated and every time they touch the patient or his bed they should wear a gown and subsequently wash their hands

Treatment of Complications—*Hæmorrhage* —This should be carefully watched for and suspected if the

patient shows any signs of collapse even if no blood has appeared at the anus He must be kept absolutely still The bed pan is best dispensed with and the patient packed round liberally with wool which is later burnt Food should be withheld for twenty four hours and even water given sparingly Above all no stimulants are to be exhibited Of drugs morphia is best of all but unfortunately it may mask perforation should that occur at the same time A platelet extract is often of value and the question of a transfusion should always be considered

Perforation requires immediate abdominal section and closure of the opening in the bowel The sooner this is performed the better the prognosis A swab should be taken to identify the predominant organism in the peritoneal exudate (which will not necessarily be the B typhosus) since if this is known further appropriate treatment may be administered

Diarrhœa is best met by withholding food for twenty four hours Especially where curds are present in the stools milk should be diluted or temporarily withheld *Meteorism* requires the rectal tube or a turpentine enema *Venous thrombosis* is treated in the same way as white leg (p 121) *Typhoid spine periostitis* etc may require surgical measures Should *cholecystitis* be prominent it should be given a chance to settle down Immediate operation is not advisable *Pneumonia nephritis carditis* etc are treated on general lines

Treatment of fæcal carriers by intestinal disinfectants such as hexamine etc is very disappointing The only effective method is to remove the gall bladder which carriers understandably enough are

usually reluctant to surrender Even this drastic procedure may fail in a certain percentage of cases in which the organism has become domiciled in the bowel To detect a fæcal carrier a mild purgative should be given and the stool should be examined as soon as possible The majority of fæcal carriers are females but urinary carriers may be of either sex Where the pelvis of the kidney is affected urinary antiseptics may be tried it is doubtful if such infection is permanent (Note that typhoid carriers may not work in any place where food or drink is prepared or sold) A quick way to detect a carrier is to examine the blood for the 'Vi' agglutinin In a true carrier this should be present even if the organisms are temporarily absent from the urine or stools

Prophylaxis—A mixed vaccine containing 500 million *E typhi* and 250 million each of *Para A* and *B* (T A B) is injected subcutaneously A week later double that quantity is introduced Immunity is developed fairly rapidly and remains at a high level for about two years Vaccination is usually an efficient preventive though it may not infallibly protect against strains of exceptional virulence

Following the injection some tenderness at the site is usual the limb should be freely used Pyrexia may be 100 F or more and general malaise may last for twenty four to forty eight hours Alcoholics may react very sharply and should abstain from alcohol for forty eight hours before and after inoculation There is also need for caution in renal inefficiency and tuberculosis

General measures of prophylaxis are of course the ensuring of efficient sewage disposal the protection of

the water supply and the attempt to ensure that no carrier has anything to do with selling or preparing food Anti fly measures should be pressed much more vigorously than they are and unfortunately there are still a great many loop holes through which infection can make its way

Control of Outbreaks —Attempt to establish a common factor between cases Concentrate on milk water shell fish and vegetables eaten raw Mean while take samples of water for chemical and bacteri ological examination chlorinate or advise boiling of all water advise boiling of milk and the avoidance of all raw vegetables and shell fish for the time being A high figure for the free and saline ammonia and especially the presence of nitrites in water should throw strong suspicion on it above all if 10 or more presumptive coli per 100 c c are found Where a given shop farm or dairy is suspected samples of stools urine and the Widal reactions of the personnel should be investigated Oysters should be bacteri ologically examined likewise lettuce cress etc Ice cream should not be forgotten Persistent and careful enquiry will usually meet with success Persons exposed may have prophylactic serum or sulphona mides with penicillin but not vaccines since the latter might well reinforce an infection as yet in the incuba tion period and thus determine a fatality

DYSENTERY

An acute infectious disease affecting chiefly the large bowel characterized by fever dehydration and

frequent loose stools which may or may not contain blood and mucus Two diseases similar in symptomatology but quite different etiologically are comprised under the term dysentery namely amœbic dysentery which occurs in tropical and subtropical countries and bacillary dysentery which in addition to the foregoing is common in temperate climates such as our own Only the latter will be considered here

Cause —The dysentery group of organisms are members of the typho coli group that is to say they are all gram negative motile and *flagellated* Individual members of the group are distinguished from one another by their fermentation reactions while each individual member may again be sub divided into several types by agglutination tests The components of the group are the *B Flexneri* the *B Shigæ* the *B Schmitzi* and the *B Sonnei* All these organisms may give rise to the clinical syndrome dysentery but whereas cases due to Flexner and especially to Shiga may be florid and severe cases due to Sonne especially in children are usually mild sometimes so mild as to escape notice Shiga infections occur in the tropics and only rarely in this country where Flexner and Sonne are predominant The organisms produce both endo and exo toxins to which anti sera have been prepared They are not however of notable value The group is also remarkable in that bacteriophage (a virus) develops in the stools and destroys the bacilli

Epidemiology —The disease is an *ingestion infection* like enteric and therefore is conveyed by food or drink which has in some way become polluted by the excreta of a person suffering from the disease or the

dejecta or fingers of a carrier The sources of infection then are much the same as enteric (p 163) with the added proviso that the dysentery organisms do not seem to possess the same hardihood or ability to survive outside the body as the E typhi and therefore outbreaks from water are very rare and from shell fish unknown But not alone may any article of food infected by a patient a carrier or an attendant act as a vehicle of spread in dysentery the fly alighting on an infected stool and then on food is believed to play a specially active part especially in warm climates It is possible that the insect itself may become infected and pass the organism in its stools at any rate for a short time and that other insects may also be concerned

Dysentery therefore may crop up sporadically or occur in epidemics depending on circumstances It is commoner in summer than in winter Sonne dysentery is especially common in children s homes and similar institutions and accounts for the fact that most notifications in this country are in persons under fifteen years of age Flexner dysentery on the other hand occurs frequently (and may become endemic) in mental hospitals and asylums where the patients are mostly adults Outbreaks in barracks and so forth have often been recorded

Pathology —The brunt of the organism s attack falls on the last few feet of the ileum and the large intestine as a whole the sigmoid and rectum being commonly singled out for special attention The mucous membrane becomes inflamed and congested to be followed by ulceration over the sites of the lymphoid patches usually lying transversely to the

mucosal folds The ulcers are covered with a layer of cells and fibrin Though often extensive in area, they are usually shallow and very rarely penetrate through the muscular coat and peritoneum to cause perforation, though they may often extend laterally to coalesce with one another and so inactivate relatively extensive areas of the bowel In any case the peritoneum over an ulcer is usually thickened As ulceration progresses dark grey or green sloughs separate which ultimately appear in the stools in the course of the separation as in enteric smart hæmorrhage may occur Oozing from the ulcers and the mucosa however is common in dysentery so that blood in any case is frequent in the stools At the same time toxin is absorbed from the bacteria at work in the bowel and toxæmia results though it is to be noted that in direct contrast to enteric the dysentery group of organisms do not invade the blood stream Usually the ulcers heal without scarring though in fulminant cases the whole mucosa may slough away

In chronic dysentery the walls of the bowel may become thick and rigid and the lumen narrowed Chronic ulceration may ensue but more often the mucosa remains coarse granular and congested

Incubation Period —Two to seven days

Segregation Period —Until two or three stools at weekly intervals are negative

Signs Symptoms and Course —This will depend largely on the type of organism At one end of the scale there are the *Sonne* infections in children in which there are often no symptoms or signs apart from slight pyrexia and a few loose stools containing a

little mucus and a few streaks of blood This may last for a couple of days to be followed by a further couple of days of compensatory constipation the child being otherwise perfectly well Occasionally there is a sharp bout of fever and symptoms may persist for a week Just as often they may subside in twenty four hours toxæmia apart from pyrexia being con spicuous by its absence In such cases it seems most unlikely that any ulceration ever occurs the bowel at most showing a patchy congestion

On the other hand in *Shiga* or very rarely in *Flexner* cases the disease may begin abruptly with high fever nausea vomiting severe bouts of colicky pains and tenesmus together with the passage of half a dozen loose yellow stools Within the next few days the stools rapidly increase in frequency and altogether lose their fæcal character consisting very often of no more than a yellowish fluid containing thick mucus and often deeply tinged with blood The colic becomes more frequent and severe the tenesmus unbearable the patient may have an almost con tinuous desire to defæcate even though usually pro ducing nothing though he may actually pass up to fifty or even a hundred stools in a day As time goes on dark green shreds of necrotic mucosa may appear in the stools and some motions may consist apparently of blood alone

Pari passu with this the patient s general condition becomes steadily more grave At first flushed and typically febrile as dehydration becomes pronounced he becomes grey and feeble the eyes sunken and the cheeks pinched In babies the fontanelle is depressed Restlessness may develop into delirium thirst may

become intense and the urine be scanty or suppressed Acidosis may appear with deep rapid respirations The blood pressure may fall and the patient pass into a state of coma and collapse with subnormal temperature and cold cyanosed extremities from which he may fail to rally though in this country death is uncommon On the other hand after one or two weeks the cramps and tenesmus may gradually disappear the stools become less frequent and gradually regain their fæcal character the temperature and pulse return to normal and the patient make a complete recovery alternatively after a certain point recovery may halt and the patient may pass into the chronic state

Chronic Dysentery is therefore a sequela of the acute disease Here the toxæmia the colic and the tenesmus are absent and the stools are reduced to six or four or fewer daily They vary considerably sometimes being formed and sometimes loose with blood and mucus as before The patient may even have a bout of constipation Although the thickened colon can be felt through the abdominal wall the organisms may not easily be cultured from the stools Recovery usually occurs though relapses are frequent and the emaciated patient is a poor subject to withstand an intercurrent infection

Complications are due to the toxin since the organism does not invade the blood Iritis irido cyclitis and arthritis are the most common the latter may persist for months Neuritis may also occur Bronchopneumonia may develop in exhausted patients An irritable colon may remain as a more or less permanent legacy only distinguishable from chronic

dysentery by the sigmoidoscope and by careful examination of the stools

Diagnosis is simple in a typical case The history clinical picture and character of the stools provide the diagnosis of dysentery microscopic examination of the fresh warm stool should be negative for *ent amœba histolytica* and culture of the mucus from the same on selective media (such as desoxycholate) should provide a growth of the bacteria which can then be identified by fermentation reactions with lactose mannitol etc In the second week of the disease agglutinins appear in the blood in many cases and may be demonstrated but the test is not regarded as very reliable Sigmoidoscopy is not to be recom mended in acute cases as it provokes hæmorrhage but in chronic cases it will reveal typical granulation patches over the site of ulcers and may even show ulceration still to be present Stools for the laboratory should as stated be fresh and quite free from dis infectant if a stool cannot be obtained a rectal swab should be sent as soon as possible as the organism may disappear rapidly from the stools

Clinically confusion may arise with food poisoning or even enteric the laboratory must form the final court of appeal In the more chronic cases ulcerative colitis or new growth may be suggested here sig moidoscopy is essential In children mucus and blood may appear in the stools in intussusception but the history and clinical picture is quite different though it should not be forgotten that a terminal intussusception may occur as a complication of dysentery itself

Prognosis—Shiga infections are much the most

dangerous and in some epidemics the mortality is high especially among the undernourished Flexner cases in this country may be quite sharp but the prognosis is very good except in infants or the aged Sonne infections are an administrative nuisance since though they demand hospitalization the children are usually perfectly well In several hundred cases one has only once seen a child that could be described as ill Adults however may have severe attacks

Treatment falls into two parts general and specific The general treatment required will depend on the type of infection Sonne cases usually require none In Flexner and especially Shiga infections the routine of the febrile state will have to be put in train Special attention should be paid to the diet which should be as nutritious as the patient can take though free of indigestible residue Fluids ought to be pushed from the outset and should dehydration become severe glucose saline should be given intravenously For the griping a hot water bottle to the abdomen is usually very helpful and a starch and opium enema or a morphine suppository may be required for the tenesmus An injection of morphia at night to secure sleep should also be considered

Of specific measures *bacteriophage* has proved a failure There are two types of *sera* to be had—antibacterial for both Shiga and Flexner and antitoxic for Shiga alone Neither has proved conspicuously successful but the earlier they are given the better the chances of success By far the most satisfactory treatment is by the sulphonamides In this country sulphaguanidine and succinyl sulphathiozole are most favoured but in the United States sulphadiazine and

sulphamerazine are preferred Whichever drug is employed it should be used in full dosage in cases of any severity remembering to keep up a free flow of alkaline urine by copious alkaline drinks In this respect sulphamerazine is the safest as it is less likely to crystallize out in the renal tubules than its related compounds

In chronic—and in Sonne—cases sulphonamides should also be employed to clear up infection Here dosage may be smaller but may have to be prolonged A nutritious diet rich in vitamins and low in roughage should be provided Liver iron and copper may be required for the anæmia which may even demand transfusion

Carriers—Dysentery carriers especially of Sonne are common What is worse they excrete the organism very intermittently so that several negative stools may precede and succeed one positive Here the rectal swab is probably of greater value than the stool Sulphonamides are useful in clearing them up but several swabs at weekly intervals should be negative before clearance is accepted Even so relapses may occur

Prophylaxis—Careful disposal of the excreta of cases and carriers is essential Anti fly measures proved most successful in the Army in the Western Desert and the advent of the D D T spray has rendered them much more simple At home our chief preoccupation should be to see that any and everybody but especially those handling food should wash their hands after using the lavatory If this could be converted into a reflex among the population generally dysentery would disappear

In tropical countries vaccines from fresh smooth strains may be employed and in a known Shiga exposure serum may be given

UNDULANT FEVER

Undulant fever (also known as *Brucellosis* Malta fever Mediterranean fever) is a disease of world wide distribution characterized by prolonged pyrexia which is usually broken up by apyrexial intervals thus giving to the temperature chart a wavy or undulating appearance

Cause —One or other of the *Brucellæ* a family of small cocco bacilli that are normally pathogenic to animals Thus the *Brucella melitensis* infects the goat and appears in the milk the *Br abortus* produces contagious abortion in the cow the *Br suis* is found in the pig and the *Br paramelitensis* in the sheep *Melitensis* and *paramelitensis* are more or less confined to the tropics and subtropics Members of the family are only to be distinguished from one another by agglutination tests

Mode of Spread —The disease is conveyed to man primarily by milk or milk products derived from infected animals either the goat or cow It may also occur among those handling infected animals or carcasses or manure—farmers slaughter house men and so on Lastly as the dung and urine of such animals are infectious contaminated water may spread the disease

Incidence and Epidemiology —The disease was for merly common among the garrisons of Malta and

Gibraltar until the practice of boiling or pasteurizing the goat's milk supplied to them was introduced Formerly believed to be confined to the Mediterranean area it is now known to occur in the Southern United States Mexico the West Indies Africa India and China In this country *abortus* fever occurs which is much milder than the *melitensis* variety Epidemics of the latter have occurred on board ships etc where infected animals were kept on board or being con

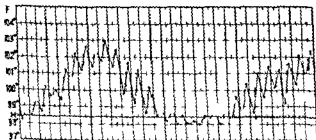


Fig 17 — Undulant Fever

veyed from one port to another otherwise the disease is endemic

The extent to which *abortus* infection occurs in this country is very uncertain Some observers believe it to be much commoner than is supposed and to be responsible for much chronic ill health Considering that about half our dairy herds are infected and that much of their milk is drunk raw (in country districts) it is astonishing that the incidence is so low and especially that it is so uncommon among women and children who proportionately consume much the greater part of our milk supply In fact adult males

preponderate forming two third of the cases and the disease is rare under the age of ten years Perhaps the frequent ingestion of small doses of infection produces immunity

Pathology —*Brucellosis* is a septicæmia and with a suitable technique the organism can be grown from the blood especially in the earlier stages The toxins produced seem to have a disproportionate effect on the heat regulating centre for considering the degree of pyrexia the patient s condition remains surprisingly good *Post mortem* in fatal cases the spleen is en larged and diffluent and the organism is found in it in great numbers The liver is also enlarged There may be congestion sometimes with ulceration of the intestinal mucosa Flattening of the cerebral con volutions has also been described Otherwise there are no naked eye changes of any particular signifi cance

The *blood* shows a leucopenia early on at a later stage there may be a secondary anæmia with a relative decrease of the granulocytes and a lymphocytic increase

Incubation Period —Uncertain usually given as from six to twenty days

Signs, Symptoms and Course —The onset of the disease may closely resemble enteric Thus there is anorexia lassitude headache pains in the limbs and often on moving the eyes constipation or occasionally diarrhœa There is a characteristic white fur on the dorsum of the tongue In severe cases there may be delirium Profuse sweating is a very frequent and prominent sign and in some cases there may even be rigors Within a few days the spleen begins to enlarge

and in time may reach the umbilicus the liver may also be enlarged but the enlargement is moderate As the disease progresses rheumatic pains in the joints may appear especially the sacro iliacs and pain in the other pressure points—the heels and the scapulæ—may also become pronounced Neuralgic pains are also common In due course the patient loses weight develops anæmia and perhaps not unnaturally becomes irritable and depressed In view of the frequency with which an apyrexial period hopefully regarded as marking the end of the disease terminates once again in a wave of pyrexia the strain on both patient and physician may become very severe

The temperature rises gradually in step ladder fashion climbing a couple of degrees in the evening and remitting a degree or so in the morning to reach a maximum of 103 or thereabouts Here with remissions it remains for several days before beginning a leisurely descent to normal or below Some days or even a week of apyrexia may follow before it commences to rise again and constitute a further undulation All told the pyrexial periods may average about a fortnight and the intermissions a week such a rhythm may persist for three months six months or even a year occasionally it may go on for even two years or longer

It must be clear that the toxæmia apart from the pyrexia is not severe otherwise it would be impossible that the patient should survive It is a curious fact that the temperature is highest usually in the afternoon (from two to four p m) rather than in the evening and that it falls during the night usually to the accompaniment of profuse sweating The

periodicity of the disease invites comparison with malaria but there is so far no comparable explanation

Complications—Chronic arthritis may develop especially, as noted, of the sacro iliac joints Neuralgic pains may persist or there may be a true sciatica In women mastitis and dysmenorrhoea may occur, though amenorrhoea is more common Pregnancy may terminate in abortion or miscarriage

Types—Several types of the disease occur The foregoing is a brief summary of a typical melitensis infection *Malignant* varieties are described in which delirium is frequent and hyperpyrexia manifest and which may readily terminate in death In *continued* cases the pyrexia is continuous and there are no apyrexial periods or undulations and sweating is insignificant or absent An *intermittent* type is notable for large daily excursions of the pyrexia with frequent mild rigors There is also an *ambulant* type where the chief sign is the daily bout of pyrexia

Abortus fever in this and other countries is generally milder and shorter usually lasting for about ten weeks though in some cases it may become exceedingly chronic A variety of types have been described as in *melitensis* cases but the general pattern of the two diseases is much the same The differences that exist are that orchitis may be prominent and that jaundice may occur in *abortus* cases Again some attacks may be characterized by severe hæmorrhages from any or all of the mucous membranes Infections sustained in laboratories may be extremely sharp

But generally speaking such developments are exceptional and there would seem to be a good many ambulant cases in which the only signs may be a

feeling of heaviness and a headache corresponding with the afternoon pyrexia In these circumstances the patient will probably continue at work without apparent ill effects

Diagnosis —There can be little doubt that in this country the disease is often missed or overlooked until the later stages In a series of nearly 500 cases the commonest mis diagnoses were typhoid or para typhoid fever influenza pulmonary tuberculosis malaria and cholecystitis in that order (Champneys) This observer remarks that if in every case of pyrexia of unknown origin in which blood is sent for culture and for a Widal test the pathologist were asked to include an agglutination test for *Brucella abortus* many more diagnoses of undulant fever would be made and there can be little doubt but that he is right The differentiation of undulant fever from the con ditions cited may be extremely difficult on clinical grounds alone unless and until the typical temperature chart makes its appearance or is recognized There are however a number of investigations which will go a very long way to establish the diagnosis provided they are made that is to say provided the practi tioner keeps the disease in mind and thinks of it in appropriate circumstances

Blood culture comes first Positive results ought to be obtained during the febrile periods and especially in the early stages but it must be made on special media in an atmosphere of 5 to 10 per cent CO_2 Such a culture if successful can form the basis of a vaccine which may later be used in treatment *Agglutination* tests are more commonly employed but unless the titre is high (1 in 1 000 or more) are considered

unreliable by some workers Unfortunately too the agglutinins do not appear in the blood for two to three weeks A macroscopic method—rocking a slide on which a dense emulsion of organisms is mixed with the suspected serum in suitable dilution resulting in snow flake agglutination—is said to be more reliable in *melitensis* infections a similar method is applicable to *abortus* There is also an intradermal test with *Brucellin* analogous to the Mantoux test in tuberculosis

Treatment and Management —Of primary importance is adequate and competent nursing care A sorbo mattress or an air or water bed is a necessity to minimize the risk of bedsores and to spare the pressure points Again owing to the profuse sweats the bed linen and nightwear need frequent changing In the second place the diet needs careful supervision Not only must it be made attractive but the vitamin and mineral content must receive special attention Wasting and anæmia are obviously bound to follow such a prolonged illness unless particular care is taken to keep up the patient s nutrition

Of specific treatment the one clear objective is the sterilization of the blood So far the effect of the antibiotics at present available has been the subject of conflicting reports which is perhaps understandable in a condition normally so variable and unpredictable in its duration and severity The sulphonamides appear to have been definitely successful in some cases Penicillin has been the subject of even more favourable reports and no doubt streptomycin when freely available will be evaluated At the moment all that one can say is that further experience is necessary

before the position is clarified In the meantime protein shock and vaccines may be employed as adjuvants

Prevention—The simplest and easiest method of prevention is the pasteurization of *all* milk Where this is impracticable as in remote country districts the bringing of milk *momentarily* to the boil offers a very satisfactory alternative Such procedures should eliminate two thirds of the infections encountered The remaining third occurring among farmers dairy hands slaughter house men etc present a more difficult problem which can be attacked in various ways One of the most obvious is that when handling infected beasts or carcasses rubber gloves should be worn and the hands washed subsequently but this would require a preliminary educational campaign among the personnel concerned

By far the most satisfactory prophylaxis of all would be the elimination of infection among our dairy herds but this presents problems of a practical and financial nature that are to say the least formidable since in the opinion of many it could only be carried out by a drastic overhaul of current farming conceptions and practice The fantastic milk yields obtained from the modern dairy cow—in other words the prolonged and intensive lactation—must surely exact a severe toll on the animal s general health and well being and on her powers of resistance to disease This is one obvious direction in which enquiry might be pursued

Prognosis—This varies considerably in different localities and according to the organisms involved In *melitensis* cases the mortality may run as high as 10 per cent but in *abortus* cases in this country it is only about one quarter of that figure Infections that

show a continuous pyrexia not uncommon in certain tropical countries, offer an especially unfavourable outlook

Acute Gastro enteritis of Children

In sharp and striking contrast with the mild Sonne dysentery so often encountered in this country is the acute gastro enteritis of children under two years in which no sufficient organism can be detected or incriminated This condition with which every physician who deals with children must sooner or later become painfully familiar, is one of the most baffling and fatal diseases of infancy and early child hood The mortality of cases may vary from twenty to thirty per cent and in any given series may well be higher still

Cause —The gastro intestinal tract of infants is still in a stage of development while at the same time it is the most actively functioning system in the in fantile economy Perhaps its development is regulated by a hormone mechanism However that may be there is no doubt that it is the most sensitive and easily deranged tissue in the child s body Any acute infection may be ushered in by vomiting and diarrhœa And furthermore it would seem that infections which would normally produce a reaction elsewhere may be expressed in the infant in terms of *gastro enteritis* Thus a *cold* in an attendant may result in vomiting and diarrhœa in the child It is worthy of note that frank tonsillitis and tonsillar diphtheria so common in older children are rare in

infants possibly because the pharyngeal lymphoid barrier has not yet developed perhaps as a result infection may the more readily pass unchallenged into the digestive tract In any case we must not exclude infection as a cause because of the absence of the common reaction in older children and adults—a frank oronasopharyngeal catarrh The very frequent finding of otitis media at post mortem shows how often infection must have passed through the nasopharynx but escaped detection

A very striking point is the rarity of the condition in children who are *wholly and entirely* fed on the breast Indeed it might be claimed that it is associated primarily with artificial feeding whatever other factors may be involved When one compares the sterility of breast milk with the invariable contamination—sometimes slight sometimes gross—of cows milk when delivered to the household this may not appear surprising In hospitals too feeding bottles may become coated with film which may defy removal and resist sterilization and consequently become heavily infected At the same time it is a mistake (in our opinion) to suppose that the infection is *always* conveyed by the feeds for there are a great many other factors which play what appears to be a significant part

One of these for example is the exposure of a child to a fresh environment The child at the breast becomes habituated to the organisms carried by the mother and probably receives antibodies to them in the milk If it be suddenly exposed to strangers and to fresh groups of organisms of which it has had no experience it is reasonable to suppose that it may not be able to resist them Experience confirms this view

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Cause—The gastro intestinal tract of infants is still in a stage of development while at the same time it is the most actively functioning system in the in fantile economy Perhaps its development is regulated by a hormone mechanism However that may be there is no doubt that it is the most sensitive and easily deranged tissue in the child's body Any acute infection may be ushered in by vomiting and diarrhœa And furthermore it would seem that infections which would normally produce a reaction elsewhere may be expressed in the infant in terms of gastro enteritis Thus a cold in an attendant may result in vomiting and diarrhœa in the child It is worthy of note that frank tonsillitis and tonsillar diphtheria so common in older children are rare in

for doing so were not generally available The decrease in the number of flies *pari passu* with the supersession of the horse by the automobile has with refrigeration contributed greatly to the eradication of this form of obvious detectable infection by known organisms which is now well on the wane On the other hand the *parenteral* infections are commonest in winter and spring i e when upper respiratory tract infection is rife and are assuming more and more importance

Bacteriologically as noted some enteral infections are due to recognized intestinal pathogens such as the dysentery and allied groups but in many others no organisms have been found though special types of streptococcus are strongly suspect In the parenteral group no definite organisms have been isolated though again the streptococcus has been accused It seems probable that many different infective agents are at work A mild enteritis is not rare during the acute stage of measles but the really dangerous variety occurs later on especially with bronchopneumonia just as in pertussis This would suggest perhaps that the enteritis is due not to the measles virus but to a secondary invader perhaps a different virus or a bacterium Swallowing of streptococci has again been advanced as causal but enteritis in scarlet fever is exceptional when very many streptococci must be swallowed Many cases seem to be associated with thrush As stated a cold or influenza in an adult may cause gastro-enteritis in a baby and from the high infectivity of certain outbreaks (especially in measles and whooping cough where coughing and sneezing occur) one is tempted to conclude that a primary nasopharyngitis due to a virus and spread

and strongly suggests that the home is much the safest environment for the baby

In theory, cases fall into two main groups—(1) the dietetic or non infectious and (2) the infectious In regard to (1) the modern view is that its importance has been much exaggerated It is easy to see how injudicious feeding may cause vomiting but is less easy to see how it may cause diarrhœa, especially over a long period and it is difficult to see how it can cause pyrexia unless in the youngest babies On the other hand it is surprising to see how children may thrive on a diet which is ostensibly quite unsuitable When they fail to do so the common sequel is not gastro enteritis but wasting The dietetic group is therefore in modern opinion gradually dwindling and the (2) infectious group reciprocally enlarging This again may be subdivided into two classes—(a) the *enteral* in which the infection is primarily conveyed to and resident in the digestive tract by the food and (b) the *parenteral* where the infection is primarily located elsewhere as in the lung or naso pharynx whence it may reach the gastro intestinal mucosa via the blood or by the swallowing of infected sputum *or it may never reach it at all* the symptoms being due to toxæmia The *enteral* group is more likely to prevail in summer when infection of food especially milk by such agents as flies is most likely to occur and when the prevailing temperature is so *high that organisms can proliferate freely therein* Specific organisms such as those of the dysentery group are often incriminated Such outbreaks used to be common and destructive before the importance of keeping milk cool was recognized, and when facilities

few days One has seen cases in which it appeared to be twenty four hours

Segregation Period—Similarly uncertain Relapse may certainly occur a week after apparent recovery

Course—The condition may set in with fever vomiting and frequent loose stools though in many cases the first sign may be a relaxed stool green from altered bile and fever or even vomiting may be absent throughout the course The stools become more and more fluid and frequent blood (and rarely mucus) may appear in them and vomiting may follow anything given by mouth Often the stools contain no fæcal matter at all The abdomen may become distended and tympanitic but very shortly dehydration appears (p 185) Sometimes it may appear with little previous enteritis It may become extreme and the collapse and toxæmia profound air hunger with *polypnœa (acidosis) supervenes and finally the child* lies immobile on his back with eyes staring fixedly at the ceiling so that death would appear to have occurred Often however a dramatic recovery ensues to be followed in a few days by a relapse This sequence of events may alternate for weeks before the issue is determined indeed there is no condition in which such surprising fluctuations occur from day to day Orange stools may often precede a fatality Recovery must be pronounced with the greatest caution a child apparently well on the mend may collapse and die in twelve hours

Diagnosis—This is self-evident as a rule But post basic meningitis may confuse so too may intussusception or even pneumococcal peritonitis

by droplet is the cause in many instances We should remember that we have no reason to believe that virus is affected or inactivated by the gastric juice The great frequency of otitis media post mortem in cases of enteritis in infants (75 per cent or more) shows that a nasopharyngitis at any rate is of very frequent occurrence the nasopharynx we know to be a favourite attacking base for many organisms whether a virus in still another instance penetrates the defence to the ultimate benefit of secondary invaders is still unsettled One thing is certain whenever a recognized intestinal pathogen is isolated from the stools the prognosis is *ipso facto* greatly improved

Within the past few years fresh light has been thrown on the condition by the advent of the sulphonamides and penicillin which shows clearly that the condition is not due to any of the many organisms susceptible to these antibiotics This diminishes the importance of the parenteral theory of causation and enhances the probability that a virus or viruses may be causal (It may be noted that in some cases the Giardia Lamblia has been incriminated)

Pathology —This is unenlightening In most cases little more than a generalized congestion of the intestinal mucosa is to be seen showing microscopically a mucous catarrh Ulceration is very rare Some bronchopneumonia is occasionally present particularly in the measles and pertussis cases Most striking as previously mentioned is the frequency of otitis media and sometimes mastoiditis In most of the fatal cases profound liver damage has been described mostly in the direction of fatty degeneration

Incubation Period —Unknown In most cases a

with a purge Thereafter the child is given nothing but frequent sips of sterile water and glucose for forty eight hours If this be tolerated well diluted milk or a patent food may be substituted gradually increasing the strength until a normal feed is being taken Should this prove unsatisfactory lactic acid milk made by adding ʒi of lactic acid drop by drop to O i of milk may be tried The resulting finer curd is said to be more digestible If nothing fluid is retained semi solids may be tried Dried apple may be very effective in babies over nine months If the stools are frothy and foul smelling indicative of fat intolerance separated milk or lactic acid separated milk may be given The greatest efforts should be made to ward off frank dehydration Giving half hourly or even more frequently sips of fluid of any kind may often prove effective if the child can be tided over the first three or four days the outlook is much better Washing out the stomach and colon with saline has been claimed to help Drugs such as tinct opii and kaolin are said to meet with success Obvious septic foci should of course be eliminated

When dehydration appears salines of normal or half normal strength may be administered often by the drip method per rectum subcutaneously intra venously or intraperitoneally A small transfusion is often of great value The oxygen tent the giving of amino acids and protein digests by mouth or intra venously salt solutions such as Darrow s or Hart mann s into the veins marrow drips—all these are procedures that have been warmly commended at one time or another Their value since they merely combat

Prognosis is almost impossible A moribund child may recover or a recovering child collapse and die suddenly The longer dehydration persists the worse the outlook A stinking seminal smell from the stools is also of evil omen The prognosis improves with every month of life Under nine months it is always uncertain

Treatment —In this condition we are dealing with a profound disturbance of the water balance of the body a subject on which our knowledge is so far scanty and inexact Growth we are apt to forget consists largely in the permanent acquisition of water and body weight again is as to four fifths a record of the amount of water we hold embodied in a complicated system of osmotic and other tensions Acids and bases metals and organic compounds all play their part in this process though the elucidation of the roles they fulfil is far from complete Thus in gastro enteritis acid is lost in vomiting and base in the stools If base is lost from the intercellular fluid the body cells will tend to shrink until the intra and extra cellular osmotic pressures regain equilibrium it is therefore only rational that salt solutions should be administered to restore the position but it must be admitted that the results are not always impressive Clearly many formidable biochemical problems have yet to be solved before such replacement therapy becomes really effective and even then we still have the problem of the cause to contend with At present we are compelled to treat symptoms hence treatment is unsatisfactory and very often disheartening

The patient should be most strictly isolated from children of a like age Many physicians commence

baby should have his own and any bottle used by a baby with enteritis is better destroyed

NOTE ON FOOD POISONING

The term food poisoning is an extremely comprehensive one stretching on the one hand (as in theory it may) from the eating of poisonous mushrooms or the drinking of arsenic in beer to the ingestion of parasites in the encysted stage such as *tænia solium* on the other In between are a large number of infections which though technically coming within the ambit of the term are by tacit consent excluded such as cholera the typhoid infections and the dysenteries In fact the term has now come to be restricted to bacterial food poisoning and indeed almost to contamination by two types of organisms—the *Salmonella* group and certain types of enterotoxin producing staphylococci—just as it was formerly believed to be due almost exclusively to the ptomaines those largely hypothetical alkaloids to whose malignant presence in meat and fish so much mischief was formerly ascribed

The *Salmonella* and the *Staphylococci* owe their dubious distinction largely to the fact that they evolve a toxin which can withstand considerable heat and therefore may be found in canned foods so that even if the process of canning has been sufficiently well carried out to destroy the organisms the toxins remain to give rise to symptoms when the food is eaten If such toxin laden food be taken on an empty stomach symptoms may come on almost at

symptoms is problematical but they have to be pressed in the absence of any specific therapy No case should ever be abandondoned as hopeless or too readily regarded as cured No disease more consistently confounds the prophets

Prophylaxis—The simplest, easiest and most obvious prophylactic is breast feeding If artificial feeding must be invoked then the greatest possible care should be taken to see that the milk is kept covered in a clean receptacle in a cool place and if it has to be kept for any length of time that it should be brought momentarily to the boil before making up the feed Indeed this is a practice that might well be indulged in as a routine

Small babies should be kept as far as possible within the family circle and never exposed unnecessarily to strangers Mothers should be warned against that pernicious practice on the part of many adults in inserting their heads under the pram hood and spraying the baby with their nasopharyngeal secretions This well intentioned tribute to the baby's charms may sometimes prove the starting point of a dangerous and perhaps fatal illness

In hospitals and institutions the most meticulous care should be taken in preparing feeds The habit of entrusting the making up of all feeds for the day to one nurse who is suitably masked and gowned and works under the strictest aseptic precautions (after which they are stored in the refrigerator until required) is to be commended No nurse or doctor with a cold should handle a baby at all and the nurse who deals with the excreta should never prepare the feeds Feeding bottles should never be interchanged Each

baby should have his own and any bottle used by a baby with enteritis is better destroyed

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once—fever vomiting diarrhœa and abdominal pain If the dose of toxin is a large one these signs may be augmented by cramps collapse and blood and mucus in the stools Even death may occur If the bacteria as well as the toxins are ingested a bacterial enteritis may later appear the initial toxæmia (which may be passing off) being then reinforced by the prolifera tion of the organism in the bowel Whereas toxin cases are short and sharp bacterial cases may go on for much longer and the patient may even develop bacteriæmia Death is more likely to occur in bac terial than in purely toxic cases but in either case it is relatively rare

In these days the preparation of food for canning and the process of canning itself are so carefully carried out that the amount of food poisoning from this source is negligible One has only to consider the enormous amount of such products consumed without mishap by the armed forces and by the civilian popu lations in the recent war to realize how extra ordinarily safe such canned food has become At the same time these foods should always be consumed as soon as possible after the can has been opened or else kept in a refrigerator for they do not keep nearly so well as food that has been freshly cooked

But fresh food when bought may be con taminated The *Salmonella* are frequently to be found as pathogens in certain animals thus the *B enteritidis* (Dublin) is met with in the cow the *B enteritidis* (Gærtner) in the rat and the *B suipestifer* in the pig They do not become domiciled in man so that the permanent carrier state in man does not occur if *Salmonella* are found in human fæces

it is as part or as a temporary sequela of an attack Meat may come from an animal that was slaughtered because it was ill or it may have (and commonly does) become contaminated during manufacture or handling such as in the making of pies brawns sausages and so on—products moreover which are often kept for several days in stock before being sold and consumed Or the meat may have been fouled by rats or mice while in store and so become infected with *B typhi murium* or as noted with *B enteritidis* (Gaertner) Lastly of course there are the flies and other insects that often infest butcher s shops or the larder at home distributing infection impartially

Certain staphylococci may as noted on occasion contaminate canned meats or fish and though perish ing themselves may leave behind a thermostable toxin which will later give rise to sudden symptoms when the can comes to be consumed But in general they are more likely to be encountered in more or less freshly prepared articles especially if these con tain milk or milk products Notorious in this respect are cream buns or pastries The *Salmonella* too may infect milk cream or ice cream—indeed either organism given the chance may pollute almost any foodstuff Note that neither of them alters the taste or smell—if a foodstuff smells sour or rotten it is no sign of pathogenic contamination and if it smells sweet it is no sign that it is safe

Food contaminated in a butcher s shop or at the pastry cook s is liable to give rise to a fairly extensive local outbreak which will therefore probably attract the attention of the local Health Authority In restaurants clubs hospitals and other institutions

however minor outbreaks are continually occurring which often pass unrecognized and which sometimes even when recognized, remain unsolved A typical history is that a number of people who have partaken of a meal in common have (with possibly one or two exceptions) been taken ill after a variable period with vomiting and diarrhœa They may or may not have been febrile as well These signs and symptoms may have lasted for a night or a day or two or even for a week depending on the source of the illness

In investigating such an outbreak there are three questions to be answered What was the organism or toxin involved? Which was the contaminated food? And who or what was the source of the contamination and how did it get in? The first question may be answered by sending fresh specimens of the stools and/or vomit to the laboratory An organism may be grown which will settle the point at once If all specimens are sterile and if the symptoms came on almost immediately it was most probably a toxin As to the second question specimens of the various foodstuffs consumed should be diligently sought if available they should be examined at once and it may be found that one or other will yield an organism Failing that by questioning any persons who have escaped and finding out what items from the menu they omitted a clue as to the identity of the offending item will be obtained Finally by examination and close questioning of the personnel engaged in preparing and handling the food it may be possible to discover the delinquent

Naturally it is a help if one knows what to look for Tea, coffee fresh vegetables that have been subjected

to prolonged boiling etc are most unlikely to be at fault Enquiry will be directed—usually too readily—to canned products or—more profitably—to food stuffs which have not been baked or boiled or roasted and especially to those which may have been prepared overnight or early in the day and left to stand for some time before serving up for unless they have been kept in a refrigerator any such dishes may well have afforded the contaminating organism the opportunity of proliferating freely More often than not such enquiries will yield information of considerable value

For instance in one such outbreak recently reported a vanilla cream was suspected as the result of a preliminary enquiry Bacteriological investigation disclosed the presence of a *Salmonella* (*typhi murium*) in the cream and also in the fæces of those affected The ingredients of the vanilla cream were next investigated separately and it was established that it was the gelatin in the cream which was at fault One member of the kitchen staff was found to have had diarrhœa some days previously without reporting it she was also found to have the *typhi murium* in her stools The remainder of the kitchen staff was negative It remained a moot point whether the suspect had contracted the infection from the gelatin or infected it herself at the time of making the cream

Two further instances may be quoted to show in what unusual ways infection may arise A woman opened a tin of soup of which she warmed and ate half putting the remainder away in the larder She had a septic finger About a week later she took out the remainder of the soup warmed it up and consumed it Within a very short time she was taken violently

ill with vomiting diarrhœa and algid collapse and died within twenty four hours Some of the remains of the soup left in the tin was investigated it was found to consist of an almost pure culture of staphylococci Doubtless these had derived from her finger and as the prevailing temperature was relatively high they had multiplied freely in a very congenial medium

In another example about one hundred soldiers engaged in manœuvres were captured by the enemy" who however, found himself compelled to retreat The prisoners were therefore warned to be ready to move at first light The cook consequently prepared a stew of bully beef and potatoes overnight for breakfast next day, and put it in a haybox to keep it warm Incidentally a few days previously the cook had developed a discharging ear which he had not reported Early next morning all the troops breakfasted off the stew with the single exception of the sergeant major who by some uncanny prescience contented himself with a modest collation of bread and jam Within six hours every man—with the exception of course of the sergeant major—had been admitted to hospital with fever vomiting and diarrhœa From both *vomitus* and fæces a staphylococcus was recovered indentical with that grown from the cooks ear Clearly the cook had infected the stew probably when putting it in the haybox thereby conferring on the organisms almost ideal conditions for proliferation

The main purpose of this note is to emphasize once again the absolute necessity for complete cleanliness on the part of all those handling food whether in our food factories our wholesale establishments our shops

or above all our kitchens The hands especially should be kept scrupulously clean Not merely should they be washed as a matter of routine whenever the individual has been to the lavatory but the public should be educated in the dangers from septic lesions or discharges as well as from fingering the nose—that handy and prolific source of organisms Soap warm water and the hand towel are the best prophylactics against food poisoning

SECTION VIII

VARIOLA VACCINIA AND VARICELLA

SMALLPOX

SMALLPOX (Variola) is an acute extremely infectious disease, characterized by severe constitutional symptoms and an outcrop on epithelial surfaces confined usually to the skin and the mucous membrane of the upper respiratory tract Formerly common throughout the world it is now rare where vaccination is practised Since vaccination is by no means universal in this country localized epidemics still occur fortunately mostly of the milder type Smallpox is the most highly infectious disease known

Cause —A filter passing virus of which two strains are believed to exist the oriental and the occidental The former causes **Variola Major**, a dangerous and disfiguring disease with a high mortality and the latter produces **Variola Minor** (*alastrim*) the morbidity and mortality from which are by comparison almost negligible Though these two forms may co exist it is not believed that metamorphosis from one to the other can occur A third form of the virus may exist in nature (cowpox) or can be artificially produced by passage of either virus through such a relatively insusceptible animal as the calf as a result of which the virus loses much of its infectivity and the power to produce a generalized eruption but still retains most of its essential antigenic constitution Hence either

form of variola will protect against the other for life and vaccination properly performed will protect against or modify both for many years

The virus is so minute as to pass a collodion filter but can be grown especially on chick embryo and subsequently centrifuged down Under the electron microscope it appears as an elongated sausage shaped body The *Paschen bodies* normally discoverable in pock fluid are probably the virus itself and the *Guarnieri bodies* to be seen in the cells of infected rabbits are probably aggregations of the virus Antibody to the virus is developed either by vaccinated persons or those who have recovered from smallpox serum from whom will precipitate the virus from extracts of the pocks or show a complement fixation reaction Neutralization *in vitro* with flocculation also occurs A source of confusion in pock fluid may be the bacteria which secondarily invade the pocks but these may be killed off by 50 per cent glycerin leaving the virus unaffected The virus resists extremely low temperatures but not boiling

Pathology —The local lesion is primarily in the nasopharynx whence the virus passes into the blood and from thence to the favoured epithelial surfaces—the skin and mucous membranes At the site of a pock the virus produces at first partial degeneration of the deeper layers of prickle cells the resultant spaces distended with exudation from the corium form tense vesicles divided up into septa by the cell wall remnants These septa are next dissolved and leucocytes invade the vesicles to form pustules Bacteria notably streptococci now secondarily invade the pustules thus accounting for the secondary fever

Post-mortem the systemic changes are those of an acute infection, showing cloudy swelling an enlarged spleen and sometimes focal necrosis in the liver In hæmorrhagic cases there are hæmorrhages into the mucous, and under the serous membranes The blood shows first a lymphocytosis but with the secondary fever, a polynucleosis

Incubation Period—Ten to fourteen days usually twelve *V minor* may go to seventeen

Segregation Period—Until all crusts have separated

Mode of Spread—By the placenta direct or indirect contact fomites droplet spray and perhaps by air currents Fomites may be very dangerous as the virus retains virulence for months

Varieties—Smallpox is described as *modified* in the resistant or recently vaccinated and *unmodified* It is also *discrete* when the pocks are separate and *confluent* when they run together—usually on the face and hands Lastly in the fatal *hæmorrhagic* type death may occur before any skin lesions appear

Course—There are in classical smallpox six stages all but the final one lasting from two to two and a half days —(1) the stage of *invasion* (2) the stage of *maculopapules* (3) the stage of *vesicles* (4) the stage of *pustules* (5) the stage of *maturation* of the pustules and (6) the stage of *crusting* In the stage of **invasion** in a mild case of *V minor* there is moderate fever, headache backache and perhaps vomiting—very suggestive of influenza which is often wrongly diagnosed In a sharp case of *V major* there is usually high fever (103 to 105° F) severe frontal headache and often agonizing pain in the back suggesting lumbago Vomiting and epigastric pain may also be

prominent and insomnia with active and dangerous delirium or perhaps severe general prostration is frequently encountered During this stage the *prodromal rashes* appear These may be erythematous or hæmorrhagic The former may be scarlatiniform morbilliform or even urticarial the latter may be purpuric or petechial Very often these rashes are confined to the *bathing drawers* area The erythematous rashes fade when the eruption proper appears but the hæmorrhagic rashes persist

The eruption proper or focal rash consists at first of tiny pin head sized macules which within a few hours become raised palpable papules firm and shotty to the touch These steadily enlarge and the walls thin out until in a couple of days they have become pearly white vesicles set on an inflamed areola of skin They are firm and flat topped often showing a tiny central depression—umbilication—and are divided up by septa like an orange so that if pricked they do not collapse As leucocytes emigrate into the vesicles these continue to enlarge to the size of half a pea the flat tops are rounded out the septa disappear and the contents become increasingly turbid so that in a further two days they are pustules hard tense and full of yellow pus The areola may disappear Secondary organisms also invade the pustules which now mature and in a further two days commence to desiccate a dark brown crust forms from beneath which a foul honey-coloured fluid may sometimes weep Four or five days later the crusts begin to separate If the true skin has not been involved healing may leave no mark, but where the lesion has lain deeper a hollow dark brown scar

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('pitting') results The crusts on the palms and soles ('seeds') are deeply buried in the skin, and may not separate for weeks They often have to be snipped out with scissors

So much for the eruption What of the general condition? With the appearance of the maculo papules the invasive fever abates and by the time the

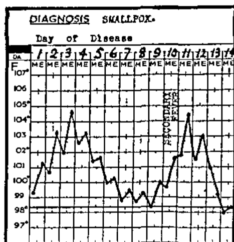


Fig 18—Smallpox

vesicles have appeared the patient is tolerably comfortable In a mild case he may even resume work As the pustules form however the fever returns—the **secondary fever** This reaches its height during the stage of maturation of the pustules—roughly from the eighth* day onwards and is usually far more

* The student often finds the time relations confusing unless he remembers that lesions on the trunk are usually about two days behind those on the face in development

severe than the fever of invasion The patient in a confluent case is then in a sorry state The face is a black honeycomb of pustules which weep a foul fetid pus so swollen generally that the features are almost obliterated He cannot see through the swollen lids and if there are as commonly pocks on the tongue pharynx or larynx the tongue may fill the buccal cavity and the unhappy patient cannot chew swallow or speak His hands are so swollen as to be useless and the itching and tension of the skin over the face palms and soles may be unbearable So much toxin is absorbed from so many points of suppuration (upwards of 10 000 in a well grown adult male) that the toxæmia is profound Often he sinks into the low muttering delirium of the typhoid state and death from exhaustion and toxæmia ensues commonly between the twelfth and the fourteenth day

Fortunately V *major* is unusual in England nowadays apart from an occasional imported case and confluent cases of V *minor* are rare Discrete eruptions seldom approach this intensity In hæmorrhagic cases which are rarer still bleeding may be profuse into the eye into the skin and from any or all of the mucous membranes and the patient may die before the outcrop appears at all leaving the diagnosis to be inferred from a history of exposure He seldom lives beyond the fifth day In contrast modified V *minor* may be so mild and the character and development of the lesions so atypical that it may easily be confused with chicken pox But the distribution of the rash remains constant The distribution of smallpox is therefore of cardinal importance The

eruption appears first on the forehead * next on the wrists later still on the ankles and last of all on the trunk and proximal limbs From the forehead it spreads down over the lower face neck and chest to the abdomen It tends to avoid the flanks From the wrists and ankles it spreads over the hands and feet and up the limbs It is profuse on the prominences but avoids the hollows in a doubtful case a pock in the axilla will tend to exclude smallpox It may attack the cornea and the mucosa of the eyes nose nasopharynx and pharynx the tongue and oro pharynx the larynx trachea and bronchi and even the rectum and vagina Wherever there has been pressure the eruption may be profuse—such as where a belt or garter has been worn From its first appear ance on the forehead until it reaches the abdomen two or more days may elapse the lesions on the abdomen are correspondingly later in development No crops ever occur The eruption is always thickest on the face forearms and hands legs and feet and thinnest on the trunk and the parts adjacent to it in a word it is *centrifugal* fleeing as the word implies from the centre to the periphery

Modified Smallpox may occur as noted in those whose vaccinal immunity is dying down All stages of the disease are likely to be milder than in the unmodified and in particular the rash tends to abort in whole or part at an early stage The stage of pustulation therefore may not develop

Variola sine eruptione may occur in those who are almost but not quite immune A transient illness

* Very rarely the rash begins on the body though the distribution is later normal This is more likely to occu in V *minor*

may develop during the second week after exposure with perhaps a few atypical skin lesions It s import ance lies in its tendency to be overlooked

Complications —These depend mostly on the site of the lesions Lesions are common on the con junctiva thus **conjunctivitis** is a frequent and early occurrence Much less frequent but far more serious is a **corneal ulcer,** which may leave a central scar and thus greatly impair vision or may even perforate thus producing panophthalmitis and subsequent loss of the eye **Laryngitis** with **œdema** of the **larynx** may de velop and provide an acute emergency perhaps necessitating tracheotomy and pocks in the trachea or bronchi may readily provoke a septic broncho pneumonia by reason of the secondary invasion which they invite

During the secondary fever **septicæmia** may easily occur leading to multiple manifestations should the patient survive In the later stages **boils** and **abscesses** readily appear and **bedsores** may be most troublesome **Erysipelas** may easily develop A demyelinating **encephalitis** is a rare event **hemiplegia** has also been described

Diagnosis —Before the eruption appears confusion may readily arise In hæmorrhagic cases the virus may be found in buccal scrapings During an epi demic, or in a patient known to have been exposed the combination of fever vomiting headache and backache should always arouse the sharpest sus picion such symptoms if coupled with an erythe matous or petechial rash confined to the bathing drawers area may be said to be diagnostic Other wise any febrile illness may be suggested most

commonly influenza or pneumonia Where vomiting and abdominal pain coincide appendicitis may be diagnosed Sometimes however the lumbar pain is so prominent that lumbago is suspected When a prodromal rash is present, scarlet fever measles enteric or even cerebro spinal fever may be simulated

As soon as the eruption proper appears the diagnosis has to be made mainly from chickenpox (p 237) mistakes are also made with *measles* papular *syphilides scabies acne lichen urticatus impetigo* or a *bromide* or *iodide* rash If it be remembered that a *localized* eruption on one or both limbs or on the face or trunk *alone* cannot be smallpox such confusion would less readily arise Where an eruption is doubtful, vaccination should be performed on or after the sixth day If it takes " the case is not smallpox Complement fixation tests with the blood and flocculation tests between the contents of the pocks and a known immune serum may also be performed An agglutination reaction is also available In *Paul s test* a rabbit s cornea is inoculated with vesicular or pustular material Two days later the animal is killed and the virus may be demonstrated in the corneal lesions Of course as soon as the electron microscope becomes generally available no doubt direct examination of nasopharyngeal swabs or vesicle fluid will be the obvious method of choice Meantime a smear from an *early* vesicle stained by Paschen s method may enable an experienced observer to recognize the virus as Paschen bodies (Van Rooyen)

Prognosis—The most important single factor is the strain of the virus the fatality rate of roughly fourteen thousand cases of *V major* in London

(1889 1904) was about 12 per cent whereas in roughly the same number of cases of *V minor* in London it was 25 per cent The next most important factor is the question of vaccination The mortality amongst the unvaccinated (from 1889 1904 in London) was four times that of the vaccinated at any time and twenty times that of those vaccinated within the previous twenty years The type of attack is also important nearly all hæmorrhagic cases die and of confluent cases of *V major* 60 per cent are fatal Finally age must be considered nearly every second child under five who contracts *V major* dies

Treatment—Recent *effective* vaccination will practically always prevent smallpox but once the disease has developed, no specific treatment is available The problem is one of alleviating symptoms averting complications and of preventing the spread of the disease

The patient must be rigidly isolated The technique of chamber nursing must be carried out with the most scrupulous fidelity Like most virus diseases smallpox is a bacteriæmia the virus circulates in the blood in the early stages and is later abundant in the pocks and subsequently in the crusts where it may lie dormant for a considerable period It is most tenacious of virulence Cotton from Virginia has carried the infection to England Fomites therefore must be disposed of with the greatest care The attendants themselves should be recently vaccinated if caught unawares vaccination during the first three days from exposure may protect and during the next three or four days will probably modify an attack.

The patient should be nursed in a darkened room if the eyes as commonly become involved and preferably on the ground floor since delirium during the invasive stages and again during the secondary fever may be very violent and the compulsion to escape may be very strong The diet should be as nourishing and as easy to swallow as possible With a copious oropharyngeal outcrop ice cream may be very acceptable An air or water bed or sorbo mattress is indispensable in severe eruptions Frequent blanket bathing or better still baths during which the patient is copiously lathered with a bland soap are very comforting and excellent therapy as well since the cleaner the skin before the pustular stage the lighter the septic invasion and when pustulation has occurred the less the toxic absorption Penicillin or a potent sulphonamide should be given as soon as the vesicles appear and should prove a useful factor in diminishing this secondary septic fever Toxic absorption and discomfort may also be lessened by snipping mature lesions or blebs evacuating the contents and dusting with an emollient antiseptic powder—e g zinc and boracic Painting with xylol has been warmly advocated The hair should be cut—it will fall out in any case if the eruption is heavy Lint soaked in water 3 parts glycerin 1 part may be applied to the outcrop generally and is very soothing The palms and soles are most relieved by fomentations Rings should be removed once the disease is recognized or they may have to be cut away with great difficulty later on The toilet of the mouth must never be neglected A steam kettle containing Tinct Benz Co ʒi to O i will keep the nasal passages

clean and is helpful should laryngitis or œdema of the glottis occur

In the crusting stage frequent baths are much appreciated and materially help in softening the crusts Olive oil is also of value and *gentle* swabbing may be employed to dislodge them *But no attempt should be made to remove any lesion until all inflammation has died down* This applies even more forcibly to the dessicated brown seeds on the palms and soles When absolutely quiet they may be snipped out with iridectomy scissors Remember that they are infectious and must be destroyed

Convalescence following a severe attack must be long and unhurried Liberal nourishment tonics and a change of air should be prescribed

Control of Outbreaks —Quarantine for eighteen days Vaccinate all discoverable contacts as soon as possible for the first five days of exposure this may very well protect and for the next five it will possibly modify but thereafter it may do harm Those exposed may be given stamped addressed postcards to notify any changes of address and all interested health authorities should be informed Surveillance should be continued for sixteen days Do not forget fomites —indirect contacts in laundries and libraries run considerable risk

PREVENTION OF SMALLPOX—VACCINATION

Active immunity to smallpox may be developed in three ways —(*a*) by an attack of the disease (*b*) by the inoculation of unmodified smallpox material (usually from mild cases)—" variolation " and (*c*) by

the inoculation of human smallpox material modified by passage through the calf, or of the material of cowpox—the agent in either case being (as far as is known) identical, and the process being known as **vaccination** The condition resulting from vaccination is known as "**vaccinia**" While the immunity which follows (*a*) or (*b*) is lifelong that from vaccination is valid only for a limited period (seven to ten years) **Variolation** has long been abandoned, though in the hands of experts it often gave excellent results The contents of a pustule were inoculated intradermally *This produced vesicles and pustules at the site of inoculation, and in addition in about ten days an attack of smallpox often very mild But however mild it was as infectious as smallpox ordinarily is and moreover occasionally proved fatal

Jenner was the first to realize that the agent of smallpox and that of cowpox, as it normally occurs in the cow were (antigenically) identical if differing in degree He observed that cowpox when contracted as commonly by dairymaid and cowherds protected against smallpox though it produced only a localized pustule (and not a generalized eruption) in the human subject It has since been established that if a calf be infected with human smallpox the resultant lesions are identical with cowpox and behave similarly when reinoculated into human subjects The cow therefore by some obscure chemistry transforms variola into vaccinia That is the basis of vaccination

Vaccinia in human subjects can consequently be produced by inoculating with (*a*) cowpox material from cowpox occurring naturally (*b*) material from calves inoculated either with smallpox or vaccinia

material or (c) material from another human subject suffering from vaccinia (This last arm to arm vaccination is now forbidden) Modern vaccination lymph is obtained from the vesicles produced on the shaved abdomens of healthy calves by the inoculation of vaccinia virus passed through rabbits The vesicles are curetted the lymph is emulsified with sterile glycerin and a trace of clove oil and stored below freezing point If approved it is issued in fine capillary tubes ready for use

To vaccinate by scarification the skin over the deltoid is carefully washed with soap and dried with a sterile towel The operator breaks off the ends of the tube and expels a drop of lymph on to the cleansed area spreading out the drop with the flat surface of a sterile scalpel blade He then scratches the skin through the film of lymph which he rubs well into the scratch * This should not be more than half an inch long and not deep enough to draw blood Officially it is recommended that only one insertion should be made The lymph is allowed to dry in the air and is then covered with a sterile dressing Above all no antiseptics must be applied or they may destroy the virus and the operation will fail For cosmetic reasons vaccination may be performed on the thigh but is prone to produce ulceration unless the limb is kept at rest

Vaccinia.—Three days after vaccination a flat red papule appears at the site of scarification By the fifth day this has expanded into a clear vesicle set on an inflamed areola On the eighth day the vesicle

* In the multiple pressure method the lymph is pressed in at 15 0 points with the flat of a Hagedorn needle.

becomes turbid, and "maturation" (probably secondary septic invasion) sets in By the tenth day it has developed into a mature pustule set on an angry red base and if the reaction is severe the arm in the neighbourhood is swollen indurated and painful, and the axillary glands are enlarged Desiccation now ensues the swelling resolves and by about the fourteenth day a thick brown scab has formed This separates in roughly a week to leave a characteristic pitted or "foveated" scar Note that in persons who are partially immune all these reactions may be accelerated

Complications —Normally the arm is painful from about the fourth day onwards and slight fever may also be present Should gross infection occur this reaction will be emphatic and erysipelas cellulitis or even septicæmia may develop as from any infected wound or abrasion Such sepsis will not hinder the development of immunity, but need not occur if an aseptic technique is properly carried out Prophylactic sulphonamide medication or penicillin should minimize it

Generalized Vaccinia is a rare condition in which in addition to the customary local developments a generalized eruption of papules vesicles and pustules is said to appear about the seventh day It would almost seem as if the virus had reverted to its old smallpox character Although occasionally reported one is bound to confess to a feeling of scepticism as to the validity of the diagnosis For if the virus really reverted to type cases should occur not singly as usually reported but in batches corresponding to the distribution of the lymph in which reversion had

occurred In one recent series of 250 000 vaccinations only three cases were reported The alteration may of course be due to the individual vaccinated and not to the virus or to the way in which vaccination is performed Should a patient first scratch the primary vesicle and then other parts of the body multiple lesions may also ensue

Encephalitis is a rare sequel to primary vaccination which does not occur in children under one year It is peculiar in that widespread demyelinization of the white fibres occurs in both brain and cord thus contrasting sharply with encephalitis lethargica Wherever possible therefore vaccination should be performed in infancy

Vaccination by injection has also been carried out the material being introduced intradermally or sub cutaneously and the puncture mark being sealed off with an antiseptic In either case indurated inflam matory swellings develop with mild constitutional symptoms but the whole reaction is much less sharp than that which usually follows the scarification method Immunity is said to be equally high but this has been questioned There is no doubt that much if not all the pustulation of ordinary vaccina tion is due not to the virus but to septic organisms and is moreover quite redundant since it does not enhance immunity to smallpox in any way The injection method should abolish sepsis and should leave no scar (This last may not be an unmixed blessing) Attempts have been made to put the matter on a still higher scientific basis by growing the virus on allantoic chick membrane and subsequently inoculating intradermally Vaccination with chick

embryo virus however performed will in any case probably supersede the calf lymph virus Vaccination may yet become as precise and uneventful as diphtheria immunization

NOTE —A certain amount of controversy in regard to the reading of vaccination results has followed from Army experience during the war In view of this it is as well to restate the position (1) No vaccination should be considered successful *unless a vesicle* has been produced (2) Never accept one failure to take as evidence of immunity (3) Vaccination *after* exposure to smallpox cannot be relied on to protect, and finally (4) two insertions are better than one and three are better than two The old four insertion technique had much to commend it

CHICKENPOX

Chickenpox (Varicella) is an acute highly infectious disease characterized by mild fever and a predominantly vesicular eruption on epithelial surfaces mainly the skin and the respiratory mucosa In contrast to smallpox the eruption appears in crops

Cause —A virus closely related to if not identical with that of zoster cases of which in adults may give rise to chickenpox in children or vice versa (The varicella virus will not grow on chick embryo thus providing a strong contrast to variola which grows freely and a point which may be of value in some cases in differential diagnosis) The converse may also occur One attack of chickenpox usually protects against chickenpox for life and both diseases protect against one another in some degree Occasionally both



PLATE XII

[illegible]

[illegible]

diseases may occur simultaneously in the same patient It hardly ought to be necessary to point out that neither disease has any relationship whatsoever with smallpox

Incidence —Any age but predominantly children under ten Infants under one are frequently attacked

Incubation Period —Fourteen to twenty one days

Segregation Period —Usually until the last scab has separated (probably from the scalp) Many authorities consider that lesions are no longer infectious when crusted In one s own experience this is not to be relied on

Mode of Spread —Direct and indirect contact fomites droplets and possibly air borne Droplets are a most important vehicle of spread

Varieties —In addition to the common type very rare *bullous* and fatal *hæmorrhagic* types have been described The so-called *gangrenous* type will be considered later

Pathology —The primary local lesion is in the naso pharynx The virus invades the blood stream and thence migrates to the chosen epithelial cells of the skin and respiratory mucosa where it comes to lie intranuclearly It attacks the superficial layers of prickle cells just beneath the horny layer producing a more rapid and complete destruction than the virus of smallpox so that the lesions are much more superficial develop more rapidly and have no septa consequently if pricked they collapse at once In addition to serum the vesicles contain the virus as elementary bodies which are agglutinable by convalescent serum (Amies)

Course —The stage of invasion is short—rarely

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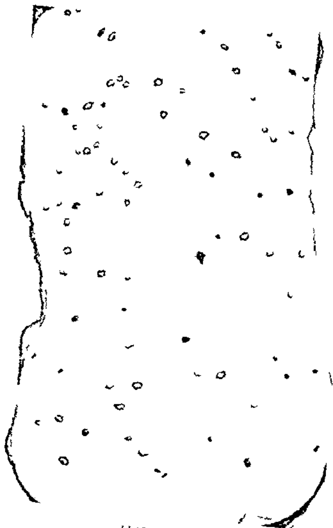


PLATE XIII

more than twelve hours and often absent There may be moderate pyrexia with mild faucial con gestion and a slightly papillated tongue occasionally accompanied by a prodromal scarlatiniform rash But frequently the first sign to be detected is a vesicle on the trunk or buttock, or occasionally the soft palate The vesicle is usually preceded by a pink macule which quickly becomes a papule but the development is often so rapid that these stages are missed even in cases under observation It is not uncommon for a child to be passed as clear at say 10 a m who by noon will show several well developed lesions Clear oval vesicles sometimes like drops of water on the skin lying with their long axes in the line of the natural body folds and often surrounded by a blotchy oval areola appear rapidly and in succession on the trunk face and limbs The eruption is always heaviest on the trunk and proximal limbs and moderate on the face The distribution which is centripetal thus reverses that of smallpox The lesions vary markedly in size and in development the largest may be the size of half a pea and the smallest that of a blanket pinhead some are fully developed but many abort The eruption itches so that rupture by scratching is a common event and as the scratching finger has not infrequently explored the nose beforehand infection of the vesicle with streptococci as well as the staphylococci is not infre quent Such vesicles as escape rupture by scratching or pressure rapidly lose their transparency and soon become turbid Within a day or two they desiccate to form a brownish crust which may not separate for a week or more Following separation unless secon

darily infected all that remains is a transient red dish brown stain Pitting is rare and even scars are small and unobtrusive though usually a few larger ones stand out (mostly on the trunk) as evidence of the attack (see Plate XIII)

About forty-eight hours after the initial eruption a fresh outcrop appears sometimes preceded by a

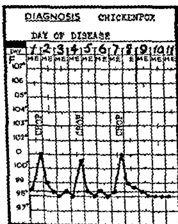


FIG 19 —Severe Chickenpox

spike of pyrexia As some of the first crop lesions have crusted by then vesicles and scabs may be seen side by side This never occurs in smallpox where the lesions on any given part of the body are all of the same age Two or three such crops may appear As a result the number of pocks may run into hundreds or even thousands though *confluent* chickenpox is exceedingly rare As with

the legs and almost never gives the distribution of varicella Impetigo vesicles sometimes confuse apart from the face they appear on sites within convenient range of the scratching finger (therefore very seldom on the back) Scabies should be excluded by the presence of the burrows

Prognosis almost invariably good

Treatment—The patient is more comfortable in bed until the crusts have separated Prevention of scratching by mittens and cardboard splints to the arms is advisable in children Baths are grateful especially at the crusting stage If irritation be severe painting with Mitman s paint (12 per cent tannic acid in collodion) is soothing and protective A menthol or calamine ointment may sometimes be substituted The crusts are best allowed to come away naturally In severe sepsis penicillin or one of the sulphonamides should be given In gangrenous cases a swab should be taken at once and diphtheria antitoxic serum or one of the sulphonamides or both should be given in conformity with the findings Glycerin and mag sulph paste locally is also of value

Control of Outbreaks—Quarantine for three weeks following a case though children may remain at school for the first ten days since during that time no infection will be disseminated Patients showing scars are exempt If further cases occur the remaining contacts had best be isolated separately else the quarantine may have to continue for months It is possible to protect by injecting 5 c c of (human) convalescent serum or 10 c c of whole blood but the production of serum in bulk has not been considered worth while

smallpox pressure or trauma may encourage a profuse localized outcrop, in a child who has recently had serum a plentiful circumscribed harvest of vesicles may appear at the injection site should this have been a limb the eruption may appear there first Vesicles on the prepuce or vagina may also occur

Complications are rare A vesicle on the larynx may lead however to a sharp laryngitis Most morbidity results from secondary infection of a vesicle or pock by the patient s finger Should his nasal or aural flora contain a really virulent streptococcus *erysipelas cellulitis*, or *gangrene* may follow Should virulent C diphtheriæ be introduced diphtheria of the skin may ensue which if unrecognized may lead to a fatal issue **Varicella gangrenosa** is due to such streptococcal or diphtheritic infection In the latter the lesions may erode through the complete thickness of the skin to expose the underlying tissue—muscle bone or even peritoneum Should a very virulent streptococcus gain entry a fatality may speedily ensue unless treatment is vigorous and very prompt In the absence of gangrene one has seen death follow infection of a pock on the cheek leading to thrombosis of the cavernous sinus **Encephalitis** is a rare sequela

Diagnosis —This is mainly from smallpox (see p 237) zoster papular urticaria drug rashes and rarely from impetigo or scabies Zoster occurs in adults along the line of a sensory nerve trunk is unilateral circumscribed and often very painful Papular urticaria appears in children under five and often erupts in nocturnal crops The vesicle is hard and leathery and set on a papule which may outlive it It favours the extensor aspects of the limbs often only

SMALLPOX AND CHICKENPOX CONTRASTED

SMALLPOX	CHICKENPOX
A severe and dangerous disease with a high mortality	A mild disease with a negligible mortality
Vaccination prevents or modifies	Vaccination has no effect
Prodromal rash often petechial and of bathing drawers distribution	Prodromal rash usually a generalized scarlatiniform erythema
Eruption begins on face, wrists and ankles spreading from face down over trunk and up limbs	Eruption begins on trunk and spreads up to the face and down limbs
Eruption comes out over a few days in one wave No crops	Every couple of days a fresh crop may appear
Vesicles and scabs never seen side by side	Vesicles and scabs often seen side by side
Lesions are set deep in the skin and feel hard they are of uniform size Vesicles may be umbilicated and divided up by septa if ruptured whole vesicle does not empty	Lesions lie on skin and feel soft Vesicles not umbilicated and there are no septa if ruptured whole vesicle empties Lesions vary greatly in size
Distribution centrifugal more lesions on limbs than trunk more on palms and forearms than on arms more on feet and legs than on thighs Prominences favoured hollows avoided	Distribution centripetal more on trunk than on face or limbs more on arms than on forearms and palms more on thighs than on legs and feet Prominences and hollows equally attacked
Toxæmic phase usually lasts two to three days	Toxæmia phase lasts for a few hours to a day

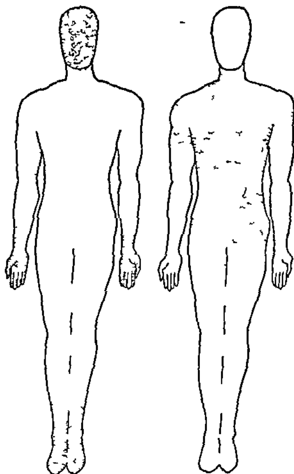


FIG 0 —The distribution of Smallpox (left) and Chickenpox (right)

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SECTION IX

INFECTIONS OF THE NERVOUS SYSTEM

Cerebro-spinal Fever

CEREBRO-SPINAL FEVER (meningococcal meningitis) is an acute infectious disease characterized by intense inflammation of the meninges and often by a rash. Many notable epidemics have occurred. This terrible disease formerly had the highest mortality of any admitted to the London Infectious Hospitals—35 per cent. of cases. One form—the post-basic meningitis of children—had a mortality of 80 per cent. or more.

Cause.—The *Neisseria meningitidis*, formerly the *diplococcus intracellularis meningitidis* of Weichselbaum (meningococcus), an organism morphologically indistinguishable from the gonococcus. Gordon distinguished four types by agglutination, types I and II predominating in this country, but it has now been established that types I and III and likewise types II, IV and others share common antigens. Much dispute has arisen as to whether the organism forms an exotoxin or not, but the question is now an academic one as serum is not nowadays used in treatment unless in very exceptional circumstances. Ferry and Fisher recovered one and produced an exotoxic anti-serum. Otherwise an anti-endotoxic serum was normally employed, produced by inoculating horses first with dead, and later with living virulent cultures

Specific type serum is more effective than a polyvalent one

Incidence and Mode of Spread —Children and young adults are predominantly attacked Over the age of forty years it is rare The disease is spread mainly by direct contact or droplet spray Consequently it is most prevalent during the cold weather especially during the first half of the year and commonly appears wherever close aggregation of susceptibles occurs—in slums among the children, or in barracks especially among country bred recruits Further subclinical attacks seem to be very common and since the disease is spread by carriers these subclinical attacks are probably major factors in the evolution of an epidemic It has been said that when the carrier rate rises to 20 per cent an epidemic will ensue In any event it is exceptional for a clinically florid case to initiate infection or to become subsequently a chronic convalescent carrier

Incubation Period —Two to four days

Segregation Period —None enforced One has never known a clinically definite case to give rise to another and patients are habitually discharged following recovery without swabs and with no ascertainable evil effects

Pathology —The point of entry is the nasopharynx Here the organism may arouse no more reaction than a mild catarrh If resistance is high it may progress no further thus producing a subclinical attack Alternatively it may effect an entry into the blood stream Such a catastrophe is usually signalized by rigors and grave constitutional symptoms In the blood stream however it may be rapidly exter

minated without further development (abortive attack) by contrast it may sometimes kill the patient before the meninges are palpably involved at all (fulminant attack) More usually it finds a congenial environment in the lateral ventricles which it reaches via the choroid plexuses wherein it multiplies and whence it invades the meninges of the brain and cord Once initiated meningitis will run its course even though (as usually) the organism disappears shortly afterwards from the blood This invariably shows a pronounced leucocytosis

Morbid Anatomy—In the most fulminant cases only those changes associated with an acute septicæmia are to be seen In less fulminant instances the choroid plexuses and the pia arachnoid show an intense inflammation the fluid in the lateral ventricles is turbid and teeming with organisms but no exudate has yet appeared In cases dying at a still later stage there is a gross purulent inflammation of all three meningeal coverings especially of the pia arachnoid Over the vertex the meninges may be smeared with pus and the interpeduncular space is often hidden by a deep mass of thickened exudate The brain substance by contrast is strikingly exempt though a few petechiæ may be seen and microscopically meningo encephalitis has been described by some observers The spinal meninges show similar changes the exudate is usually thickest on the dorsal *surface of the lumbar cord Both cranial and* spinal nerves are often invested by exudate but the axons are seldom injured In the *chronic* (hydrocephalic) stage there is gross distension of the lateral ventricles with the basal exudate in various stages of

organization In the primarily chronic type that occurs in infants the exudate is often limited to the posterior part of the base of the brain—*post basic* meningitis

Varieties —From the foregoing it can be seen that many gradations in severity may occur but we may distinguish six main types —(1) the *fulminant* type (2) the *ordinary* type (3) the *abortive* type (4) the *chronic* type and (5) the subclinical *ambulant* type Finally there is (6) the *post basic* meningitis of infants

Course —The invasion is abrupt with fever perhaps rigors headache vomiting and intolerance to light and sound In children convulsions with or without abdominal pain vomiting and diarrhœa may occur When the organisms invade the lateral ventricles and meninges the headache becomes intolerable and as the cerebro spinal fluid is secreted in rapidly increasing quantity the intracranial pressure rises steeply Thus in addition to the toxæmia signs of meningeal irritation rapidly appear at first to colour but soon to dominate the picture Earliest of these is **head retraction,** *produced by tonic spasm of the neck* muscles this spasm soon involves the erectors of the spine resulting in **opisthotonos** The back is arched with the convexity forwards grossly exaggerating the normal lumbar curve and the flexors of the lower limbs are contracted so that the patient if placed on his back would rest on his head and heels He usually lies however on his side with arms flexed and abdomen retracted facing away from the light He is stuporous resistive and intolerant of attention and as soon as a disturbing stimulus is withdrawn at

once retreats to his refuge under the bedclothes His expression is agonized and frowning because of the intensity of the headache the pain of which is persistent and often unbearable He remains sleepless for days on end and soon becomes exhausted On examination his pupils are dilated but equal rarely there is a squint The lips often show a profuse outcrop of herpes Nasal irritation is often a prominent sign he continually rubs or picks at the nose even to the point of producing ulceration The urine may be copious and contain albumin though retention may as often occur The tendon jerks are often brisk but the abdominals are usually absent A notable feature is the pulse which is steady hard and (relatively) slow

The **rash** to which the old name of spotted fever was due is in sporadic cases usually absent In epidemics a rash is much more common It appears on the first or second day It may take the form of a discrete or confluent macular erythema or an urticaria but much more commonly it is hæmorrhagic and most often then **petechial**—hence the term spotted fever The rash may cover the entire trunk and limbs In the severer cases ecchymoses ensue they indicate a bad prognosis

In the absence of treatment and indeed too often in spite of it all the foregoing manifestations may increase in severity In children especially retraction may pull the head back almost between the shoulders and convulsions may rarely occur Wasting is excessive and rapid The mouth becomes dry and foul the tongue fissured and the lips covered with sordes mingling with the crusting herpetic vesicles Violent

delirium with incontinence is common But though the signs are those of extreme toxæmia pyrexia is by no means pronounced The temperature may indeed fluctuate widely from subnormal to even as much as 105 F Neither owing to the intracranial pressure does the pulse rate match the severity of the toxæmia It may be abnormally slow The respirations may be irregular and in the terminal stages very shallow, or of the Cheyne Stokes type Death is usually preceded by stupor deepening imperceptibly into coma often within a week or ten days of the onset

On the other hand after one two or more weeks recovery may ensue though commonly chequered in its course by minor setbacks Slowly the mind may clear the headache abate the retraction lessen the tongue clean and the appetite return Even though emaciation may have been advanced tardy but complete recovery without noticeable sequelæ may take place The patient is often unsteady on his legs sometimes spastic occasionally ataxic and he may complain of vague pains in his legs and elsewhere His headache may occasionally return but on the whole he is none the worse for his terrible ordeal

In a certain number of cases however the acute symptoms abate but do not disappear This is the chronic form The retraction in particular may persist and the emaciation become progressive and extreme Bedsores are the rule and incontinence is usual Even now arrest with complete ultimate recovery may follow but in most cases a developing papillœdema shows that the foramina of Magendie—and perhaps of Luschka—are blocked by organized exudate and that hydrocephalus is supervening In

this event death from increased intracranial pressure acting on the vital medullary centres is the common sequel and if recovery does occur the patient may be left permanently crippled deaf, or blind

In the **fulminant** type death may occur during the septicæmic stage in as little as twelve hours Here following the initial rigors headache and vomiting *copious hæmorrhagic rashes* erupt *usually ecchymotic* but sometimes petechial Delirium and coma are swiftly followed by death, without discoverable signs of meningeal involvement which indeed has not had time to occur Diagnosis in such cases can only be made by culture from the blood Post mortem such cases show hæmorrhages into the suprarenals—the Waterhouse Friderichsen syndrome Between this and the ordinary form, intermediate cases may occur in which a brief *meningeal stage* develops shortly before death occurs In the **abortive** type signs and symptoms of an attack appear but soon pass off

Post-basic meningitis is a form of meningococcal meningitis occurring mostly in infants and children in which the process is confined mainly to the lateral ventricles and the base of the brain While it often starts abruptly with fever vomiting and convulsions the condition soon becomes afebrile The physician is therefore confronted with a chronic condition marked by extreme retraction and intense opisthotonos so much so that the body may almost form a loop with the occiput and buttocks in apposition (The gun hammer position) The limbs are often rigid Vomiting and a rapidly developing marasmus are prominent features though the child is stuporous it will take its feeds but without benefit

Central blindness is the rule Hydrocephalus with gross enlargement of the cranium is almost invariable and death in coma usually supervenes in a month or more from the beginning In the few cases which recover permanent deafness and blindness are very common Mental deficiency may follow

Complications—**Hydrocephalus** has already been dealt with it is much the most important **Papill-œdema** with permanent blindness may follow hydro cephalus An early complication is **deafness** often bilateral and often permanent due to meningitic involvement of the internal ear or of the nerve **Otitis media** also occurs But excepting the sixth and occasionally the auditory division of the eighth paralysis of the cranial nerves is surprisingly uncom mon in this condition Following the septicæmic stage **arthritis** is not infrequent but **orchitis pneumonia, endo-** and **peri-carditis** are rare A most troublesome sequela may be a **psychosis** similar to that which may follow head injury it usually clears up in time and permanent mental impairment is exceptional unless following hydrocephalus The latter may also produce a hemi or di plegia

Diagnosis—During the septicæmic stage this can only be made by blood culture though a blood count will show from 15 000 to 40 000 white cells (poly nuclears predominating) thus excluding enteric In epidemic times a purpuric eruption should always arouse suspicion here a blood count should similarly exclude hæmorrhagic smallpox or fulminant purpura and show that a septic organism is at work In the vast majority of cases retraction is present very early a hand placed on the neck muscles will detect the

tonic spasm and the patient will be unable to flex the chin on the chest Two further simple signs of meningeal irritation are —(a) Kernig s if the thigh be flexed on the abdomen the leg cannot be fully extended on the thigh, and (b) Brudzinski s if the chin be forcibly flexed on the chest the lower limb will flex at the hip and knee These signs may be *absent in infants and small children, in whom however* drowsiness and retraction will be present, which should always arouse suspicion Indeed *whenever retraction in any degree is present* unless obviously due to enlarged cervical glands, *lumbar puncture should be performed at once*

Lumbar puncture is such a well known procedure that it need not be described here It is however important to remind the student that when the needle is *in situ* he should *control the flow which must be in single drops* otherwise meningeal hæmorrhage may occur Drainage is continued until the flow is normal (The rate varies with the size of cannula employed) The fluid should be collected in a sterile bottle, cultures are made part is reserved for chemical examination and part for immediate microscopical investigation If intrathecal penicillin is contemplated it can now be administered

Dry puncture is not infrequent where many punctures have previously been performed the needle being blocked by organized blood clot exudate etc In such cases a space higher up may be employed or cistern puncture carried out

Cistern Puncture —The needle must be calibrated in centimetres It is inserted just above the spine of the axis, and directed forwards and upwards in the

direction of a line joining the glabella and the external auditory meatus Fluid should be struck at a depth of about 3 cm in a child and 5 cm in an adult The stylet should be removed a little short of these distances in either case if no fluid appears it is reinserted and the needle cautiously advanced a further ½ to 1 cm when the stylet is removed again The characteristic tightly gripped feel as the needle pierces the dura is readily recognized Hæmorrhage is more likely in this than in spinal puncture It is usually due to injury to a vein or to release of pressure rarely to injury to the medulla which with care is a remote contingency unless in hydrocephalus when the brain stem has been pushed downwards by pressure

In babies in whom the anterior fontanelle is patent ventricular puncture may be employed The needle is inserted at the most lateral point and directed downwards and slightly inwards for 4 or 4 5 cm when the ventricle should be reached

The appearance of the fluid is characteristic It is opalescent or frankly turbid and under high pressure Should the rise in pressure be doubtful a manometer may be employed to obtain a definite reading On standing or centrifuging a thick deposit of cells and organisms comes down the latter are usually readily identified in a stained smear The conditions causing confusion are set out in the accompanying table they may only be separable by analysis of the fluid Clinically the various forms of purulent meningitis (whether due to the meningococcus pneumococcus streptococcus or rarely to the typhoid or Pfeiffer bacillus) are indistinguishable from one another * and

* Unless a rash is present.

CEREBRO SPINAL FLUID

N = normal O = absent. + = increased − = diminished

Fluid	*Appearance*	*Appearance on standing*	*Pressure*	*Cells per cm*	*Organisms*	*Protein mgms %*	*Glucose mgms %*	*Chlorides mgms %*
Normal	Clear	Clear	110–130 mms water	0–5 lymphocytes.	O	·5	50–80	5–750
Meningism	Clear	Clear	+ to + +	N	O	N	N	N
Benign lymphocytic Meningitis	Clear	Fibrin web	N to + +	Lymphos + +	O	+	N	N
Epidemic Encephalitis	Clear or faintly turbid	Clear	N to + +	Lymphos + +	O	Slightly + or N	Slightly + or N	N

Poliomyelitis	Clear or faintly turbid	Fibrin web	+ to + +	Polys early then Lymphos + +	O	Slightly +	N	N
Tuberculous Meningitis	Clear or faintly turbid	Fibrin web	+ to + +	Polys early then Lymphos + +	+	+	—	600 —
Syphilitic Meningitis	Clear	Fibrin web	+	Lymphos + +	O	+	N	N
Purulent Meningitis	Turbid	Deposit	+ + +	Polynuclears + +	+ +	+ +	—	7 0 —
Subarachnoid Hæmorrhage	Bloody brown or yellow	Deposit (blood cells)	+ to + +	Variable (blood cells)	O	+ to + + +	N	N

DIFFERENTIAL DIAGNOSIS OF A CASE SHOWING SIGNS OF MENINGEAL IRRITATION

CEREBRO SPINAL FLUID

N = normal *O = absent* + = increased. − = diminished

Fluid	*Appearance*	*Appearance on standing*	*Pressure*	*Cells per cm*	*Organisms*	*Protein mgms %*	*Glucose mgms %*	*Chlorides mgms %*
Normal	Clear	Clear	110–130 mms. water	0– lymphocytes	O	25	50–80	725–750
Meningism	Clear	Clear	+ to + +	N	O	N	N	N
Benign lymphocytic Meningitis	Clear	Fibrin web	N to + +	Lymphos + +	O	+	N	N
Epidemic Encephalitis	Clear or faintly turbid	Clear	N to + +	Lymphos + +	O	Slightly + or N	Slightly + or N	N

ministration will usually suffice but the dosage should be heavy—there is no room here for half measures

Apart from the sulphonamides which deal with the organisms the indications are to relieve toxæmia reduce intracranial pressure and maintain the general nutrition The administration of serum intrathecally has happily been abandoned but in a severely toxic case serum *intravenously* may sometimes reduce toxæmia considerably Lumbar puncture to relieve intracranial pressure is now very rarely performed it is surprising how once the infection has been mastered pressure takes care of itself The maintenance of the patient s general condition is again no longer a great difficulty in these days since the issue is usually decided one way or another within a week though formerly it represented a formidable problem Again with chemotherapy the chronic stage of the disease seems to have disappeared

Penicillin in meningitis to be effective must be given intrathecally and is therefore at a disadvantage compared with the sulphonamides in meningococcal and streptococcal cases But in pneumo or staphylococcal infections penicillin is the only hope and has proved very successful It may also be required in meningo and streptococcal cases should sulphonamide resistant forms of these organisms be encountered The dosage varies with the size and age of the patient it is given intrathecally 5 000 units daily should suffice It is of particular value in children under one year

Sequelæ—Various permanent palsies have been reported but the most important sequelæ are deafness

often the tuberculous form may give rise to confusion. The latter runs a less acute course, ocular paralyses are much commoner and if primary it is often preceded by a period of mental irritability and depression. The ears should always be examined in meningitis. There is, however, little point in labouring the clinical differentiation since the last word rests inevitably with the fluid.

Prognosis.—This is worse over forty years and very bad in infants. In a case under treatment an increase in the sugar content is a hopeful sign. A good free flow of a gradually clearing fluid (especially with general improvement) is also of good omen. An increasingly turbid fluid is of bad prognosis, as is also the occurrence of a block whether cerebral or spinal. When block occurs *Queckenstedt's sign*—an increase in the flow of fluid through the lumbar puncture needle when both jugulars are manually compressed—will be absent. In general prognosis must always be guarded.

Treatment has been revolutionized by the introduction of the sulphonamides to which fortunately the meningococcus is highly susceptible with the result that the prognosis in all but fulminating cases has been improved out of all recognition. Given in full doses* to a case of ordinary severity a distinct improvement should be noted in two to three days and the patient should be normal again in less than a week. Even in fulminating cases there is some hope if treatment can be begun at once: the loading dose should be the highest possible and be given intravenously repeated if necessary. In ordinary cases oral ad

* At least 1 gramme per stone of body weight per day

much favour in this country so far *Carriers* also respond well to small doses of sulphonamides

Meningism is a condition frequently confused with meningitis in which as a result of toxæmia or some times bacteræmia signs of meningeal irritation appear *On lumbar puncture however the fluid is normal though showing an increase of pressure* Head re traction and a positive Kernig s sign are present It may arise from suppuration close to the meninges as in mastoiditis or sinusitis or in general infections such as enteric smallpox or influenza Upper lobe con solidation in pneumonia in babies and small children is a most frequent cause and poliomyelitis may occasion extreme difficulty in diagnosis Even rheu matic fever (if the neck is painful and stiff) may arouse suspicion as may grossly enlarged acutely inflamed cervical glands but here the fluid will show a normal pressure Encephalitis may also cause confusion In diagnosis lumbar puncture is essential and conclusive apart from such confirmatory signs of the masquerad ing condition as may appear or be elicited Once meningeal signs have appeared in meningitis the fluid will show *some* suggestive abnormality (In cases suspected of meningitis intrathecal serum should never be given as apart from anything else the resulting outflow of polynuclear cells will render subsequent examination of the fluid most misleading)

POLIOMYELITIS

Poliomyelitis is an acute infection of the nervous system involving chiefly the grey matter of the brain

and changes in the eye grounds and optic nerve Psychosis has already been mentioned

Chronic meningococcal septicæmia—Some cases have been reported in which meningococci were present in the blood for quite long periods, though there were no signs of meningitis and indeed the clinical disturbance was very slight In others there were rigors rashes splinter hæmorrhages in the nails erythema nodosum etc Clinically the condition may be extremely puzzling but blood culture is decisive

Prophylaxis—Immunization with the exotoxin and with vaccines has been carried out and good results have been claimed but since the disease is relatively uncommon and spread is in any case somewhat unpredictable its value is difficult to assess It is not in general use

Control of Outbreaks—This is essentially a matter of the detection and segregation of carriers and those undergoing subclinical attacks A long curved (West s) swab of the nasopharynx is taken, plated out and incubated If positive the delinquent is segregated Abundant fresh air and the irrigation or swabbing of the nasopharynx with antiseptics may clear him up failing which (with his consent) surgical measures to correct unhealthy conditions may be taken Segregation of carriers is however a lengthy and complicated matter Immediate measures to reduce overcrowding and opportunities for spread may often give very satisfactory results The American Army authorities have reported very good results from giving small doses of sulphonamides grms i to ii daily to *all* personnel over a period of a couple of weeks though this is not a method that has found

six months cases are very rare The older the patient the higher the mortality since the more common the bulbar type of the disease Males are more susceptible than females blondes than brunettes and the rich than the poor As children of one year are very susceptible while the immunity of adults is high subclinical attacks would appear to be common Isolated communities usually suffer severely Evidence has been adduced to show that there is an inherited susceptibility to the disease a fact hard to reconcile with the observation that more than one case per family is rare

Incubation Period —This would seem to depend on how the disease is contracted (see below) An average figure would seem to be twelve days

Segregation Period —None usually enforced in this country Although many cases have been unwittingly nursed in general hospitals with no special precautions instances of spread are said to be extremely rare The virus may appear in the fæces for eight weeks or longer

Pathology —The traditional view confidently held until a few years ago was that the disease was a droplet infection like measles or influenza The virus having been implanted in the nose made it s way to the C N S via the axons of the olfactory nerves whence it proceeded to the thalamus and subsequently by way of the spino thalamic tract to the lumbar enlargement of the cord where it turned forwards to attack the anterior horn cells (Why it should ignore the anterior horn cells of the cervical or dorsal cord so often was never made clear)

The second school believes that the condition is an

and spinal cord as a result of which paralysis *may* or *may not* develop Epidemics occur especially at harvest time which vary greatly in severity some may show a death rate of anything up to 40 per cent North America and Australasia have suffered severely In England the disease is relatively uncommon One attack usually confers lifelong immunity

Cause —One of the few points of common agreement in this disease (almost every other feature of which is the subject of acute controversy) is that the cause is a filterable virus usually demonstrable in the fæces and sometimes in the naso pharyngeal washings of cases in the invasive stage and in the substance of the brain and cord of monkeys in whom the disease has been reproduced Reproduction may be effected by intraperitoneal intracerebral or intranasal instillations of such washings or of emulsions of nervous tissue from fatal examples of the disease The globoid bodies grown by Noguchi and Flexner may possibly represent aggregations of the virus which is itself ultramicroscopic with the ordinary microscope though by the electron microscope it can be quite easily seen Note that it is apparently absent from the cerebro spinal fluid It resists desiccation sunlight moderate heat and weak chemicals and may persist in milk or water for a month or more It cannot be grown on chick embryo It exhibits considerable variations in virulence

Mode of Spread —This is greatly in dispute See below

Incidence —The disease may occur at any age though children predominate (The late President Roosevelt was attacked by it in his thirties) Under

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the fæces contain the virus more often than the pharynx

But whatever doubt exists as to how the virus enters the cord there is little doubt as to how it behaves once it gets there Apparently it first enters the posterior root ganglia hence the initial hyper æsthesia Thence it passes to the posterior horns Here it may die out (*abortive* attack) but too often it proceeds to the anterior horn cells where it exerts an extremely destructive effect From its point of entry it may pass upwards or downwards in the cord to (in theory) an unlimited extent but usually it remains localized to that segment of cord first attacked

Post mortem —In fatal cases there is intense con gestion of the meninges with gross œdema of the cord which when cut bellies out to overlap the severed membranes Microscopically many of the spinal anterior horn cells have been completely destroyed and many others show evidence of partial disintegration Marked congestion of the blood vessels with perivascular round cell cuffing and scattered petechial hæmorrhages are also prominent features The inflammation though most intense in the anterior horn is by no means confined to it the posterior horn cells Clarke s column and the posterior root ganglia are hardly less involved thus accounting for the marked hyperæsthesia so common in this disease Not even the sympathetic ganglia escape The prevalent œdema may produce wide-spread if [illegible] es [illegible] its pressure on cells remote from th[illegible] ion and may also compress [illegible] ucing equi vocal sig[illegible]

ingestion infection like enteric The virus having been swallowed in contaminated food reaches the gut whence it makes its way through the splanchnic nerves to the lumbar cord During ingestion it contaminates the oropharynx, where it may excite a catarrh, and whence it may also turn backwards to the pons medulla and cervical cord This is the more likely to happen if the tonsils have been removed an observation that corresponds with the clinical fact that bulbar forms of the disease are commoner in older children who have undergone tonsillectomy

A third school believes that following a preliminary droplet infection the virus passes into the blood and so to the nervous system Lastly there are those who believe that infection may take place either by droplet or ingestion as the case may be If by droplet there is a preliminary catarrh following which the virus may pass back to the bulb alternatively being swallowed in the sputum it may reach the intestine the splanchnics and thus the lumbar cord If this were so clearly there would be a discrepancy in the observed—or estimated—incubation periods which might explain to some extent the discrepancies reported

The question however still remains an open one though the consensus of American opinion favours the ingestion hypothesis One fact which militates strongly against the droplet theory is that an outbreak may occur in a town A fifty miles away from town B but connected with it by road and rail and yet town B may escape the disease entirely This would surely be an impossibility if droplet were the normal mode of spread as for instance in influenza In any event

into preparalytic and paralytic stages *either of which may be absent* Thus a child may go to bed apparently normal to awake in the morning with paralysis On the other hand he may become sharply ill with all the invasive signs of a severe attack and yet never progress to paralysis (though developing immunity) These latter cases are described as *abortive* and in epidemics may be extremely common In its most complete form the preparalytic period may be marked by two febrile bouts (separated by an interval of three or four days during which the child seems normal) before paralysis occurs This is the double hump or dromedary * chart Such cases are rare The common picture is that the child is taken suddenly ill with fever vomiting and cramping abdominal pains Constipation or diarrhoea may be present Epistaxis is common and coryza with pharyngeal congestion is the rule Herpetic morbilliform or scarlatiniform prodromal rashes may appear Generalized hyperæsthesia is an early and prominent symptom the child though drowsy is irritable apprehensive and hyperæsthetic and dreads handling which may provoke exquisite pain though indeed pain may be pronounced even at rest (neuritic type) Tremor of the affected muscles with sweating local or general is frequently noted The spine is tender and the child cannot be persuaded to flex it he may thus resist flexion of the neck to a degree indistinguishable from the neck rigidity of meningitis Most characteristic is the "tripod" sign When placed in the sitting posture the child assumes the

* This term is unfortunate since the animal has, in fact only one hump

Some writers describe auxiliary changes throughout the body—diffluent spleen cloudy swelling of the liver and kidneys and so on—highly suggestive either of a profound toxæmia or of a frank virid æmia '

The **cerebro-spinal fluid** should be examined while fresh It is increased under high pressure and typically 'ground glass" in appearance The fine web so characteristic of tuberculous meningitis may appear on standing and thus prove deceptive to the naked eye The cell content is high mostly mono nuclears and both the albumin and globulin are increased as may be the sugar (see p 248) The chlorides are normal The blood usually shows a polynucleosis

Varieties —The disease may be classified anatomic ally depending on whether the brunt of the infection falls on the cranial nerve nuclei of the pons and medulla—the **brain stem** type, or as in most cases, on the anterior horn cells of the spinal cord—the **spinal** type **Mixed** types commonly occur An *encephalitic* (Strümpell) type was formerly described characterized by convulsions coma a high mortality and destruction of the upper motor neurones with a resultant spastic paralysis but as it is impossible to produce experimentally most authorities nowadays brusquely deny that this is a form of poliomyelitis at all and regard it as a quite different disease In any event whatever the series most cases are of the spinal type and the virus betrays a constant bias for the cells of the lumbar enlargement

Course —This may be extremely erratic consider ably complicating description It is usually divided

exists between the changes in the fluid the severity of the invasive symptoms and the extent of subsequent paralysis Statistics however show that whatever the original distribution in 80 per cent of cases the legs are residually involved



Fig 21 —Poliomyelitis The tripod position

Death is very rare in purely spinal forms but in the bulbar varieties is always to be feared Since these comprise at least 20 per cent of any epidemic and often considerably more poliomyelitis may prove a very lethal disease It is one of the rare conditions of childhood in which the older the patient the worse the prognosis since as noted bulbar forms

tripod position i e the back held rigidly straight and the trunk leaning backwards against the arms which are braced behind in support If asked to fold his arms or kiss his knees he quickly abandons the attempt In bulbar cases a squint facial asymmetry or diffi culty *in speech or swallowing may be apparent, and in* severe cases meningitis may be as noted, very strongly suggested The tendon jerks are early exaggerated and *unequal* the abdominals are usually absent

The preparalytic stage may occupy anything from twelve hours to a week In most cases paralysis supervenes explosively on the first or second day following which defervescence by lysis occurs The paralysis progresses rapidly to a maximum in about six hours though rarely it may continue to spread for twenty four hours or longer (often with fatal results) Exceptionally relapse may occur

The paralysis which is flaccid results in complete loss of voluntary movement in the affected muscles which show the reaction of degeneration and loss of reflexes Since much of the initial paralysis is due to pressure of the œdema on structurally sound cells it is more extensive at first sight than it ultimately proves to be As the œdema subsides recovery of these sound cells sets in usually in about a week *Recovery in partially damaged cells follows and by* the end of six months has occurred in all neurones except those mortally injured Muscles which show a return of excitability within that time will therefore recover *with suitable treatment* in the remainder the muscle its tendon and even the attached bone show a progressive involution growth ceases and consider able *deformity results No prognostic relationship*

the fluid is normal as a rule In rheumatism influenza and scurvy, both fluid and reflexes are normal Landry s syndrome may offer extreme difficulty but the combination of a polynucleosis in the blood with a lymphocytosis in the fluid followed by an atrophic paralysis is unique and exclusive to poliomyelitis

Treatment —At this point the fires of controversy which smoulder wherever the subject of poliomyelitis is touched flare up anew It is generally agreed that confinement to bed and the customary management of the febrile state are indicated that lumbar puncture is of therapeutic as well as diagnostic value and that analgesics should be administered as required Furthermore as the virus is present in the nasopharynx and is excreted in the fæces and urine strict isolation and disinfection of the excreta should be carried out Convalescent serum is nowadays condemned as useless Atropin and nasal feeding in pharyngeal paralysis with mechanical respiration when required are measures too obviously beneficial to invite or suffer attack it is around the treatment of the paralysed muscles that the conflict rages

Briefly the orthodox method prescribes complete rest of the affected part during the acute stage—i e usually for about six weeks Harmful overstretching of a paralysed muscle by an unopposed antagonist is prevented by retaining the limb in a state of physiological rest with light celluloid splints or plaster shells When muscle tenderness is no longer apparent radiant heat massage passive movements and re education exercises (often carried out in warm water to promote the circulation and defeat gravity) are cautiously and increasingly employed Where one

are commonest in children over ten years Any cranial nerve may be attacked though the process seldom extends above the sixth most often it is the facial that is affected Death when it occurs is due to paralysis of the respiratory muscles This is usually preceded by palatal and pharyngeal involvement so that regurgitation of fluids through the nose and coughing and spluttering on drinking are early signs just as in diphtheria Failing a respirator death may ensue as early as the first day and even with its help may result months later from pneumonia The prognosis in the bulbar form is not *however one of unrelieved gloom since if the patient* survives no residual palsies ever occur As for the danger to life anxiety must be felt if the pyrexia is more than 102° F, and of course if the pulse or breathing becomes irregular

Complications —Apart from trophic changes—bed sores ulcers on the affected limbs and so on the only one of practical importance is the pneumonia that so often follows respiratory paralysis

Diagnosis during an epidemic may be easy but in *sporadic cases may be extremely difficult* There are few pædiatricians of experience who have not been discomfited at one time or another by this disease The symptoms may suggest encephalitis meningism or meningitis of any kind or where hyperæsthesia is pronounced polyneuritis acute rheumatism influenza or even scurvy In encephalitis the frequency of ocular palsies and the subsequent divergent course of the two conditions should assist The fluid should separate both meningitis and meningism Polyneuritis is usually symmetrical, shows sensory changes and

for it though some measures are of undoubted value Compromise has been suggested From the whole controversy one fact emerges clearly however The practitioner if he is wise as soon as he has verified the diagnosis will refer the case to a competent orthopaedic surgeon since in the treatment of this disease there is no room for amateurs

Prophylaxis —During an epidemic the avoidance of crowds especially indoors would seem to be advisable Children should be kept away from others as far as possible indeed the fewer people they encounter the better Food should be carefully supervised milk and water should be boiled and nothing eaten raw Public Health authorities do not advise closure of residential schools since while the individual may thereby be advantaged the general dissemination of infection which might follow could easily react adversely on the community Parents however often force their hands

The problem confronting a Public Health authority could not well be more difficult The fact is that we do not really know how the disease is spread and until we do our measures can hardly rise much above a series of pious gestures Until the electron microscope is a common item of equipment the detection of carriers (if carriers *do* play a vital part in spread) must remain a most elaborate and cumbersome procedure The strict disposal of excreta the destruction of flies and other insects the wearing of masks and so on are measures that will obviously do no harm and may do good But until the mechanism of dissemination has been finally and satisfactorily worked out it is obviously impossible to lay down any hard and fast

muscle of a group with common action is involved electrical stimulation may be included but voluntary exercises intelligently controlled form the basis of treatment, which should be kept up for years Fatigue which retards recovery should always be avoided Progress should preferably be recorded on muscle charts Only when no further recovery is likely should the patient be allowed up—which may mean confinement to bed for a year or even longer *It is important to note that a certain quantum of improvement can always be secured by treatment whether given immediately or even after the lapse of years*

Diametrically opposed to this is the Kenny method which decrees that muscle pain is due not to inflamed posterior root ganglia but to spasm circulatory stasis and thus anoxia Fomentations or diathermy are applied to the painful muscles which are passively exercised from the outset in spite of pain When defervescence has occurred alternate hot and cold sprays in the bath are employed to restore circulation massage being avoided Passive and active movements are practised from the outset carefully avoiding fatigue and at no time is the limb immobilized The patient is encouraged to place the feet to the ground and gradually to take the body weight as soon as possible Great emphasis is placed on helping the patient to overcome inhibition and to overcome the psychological barriers to recovery This method has its zealous supporters and equally determined opponents whatever its merits they have certainly been pressed by methods scarcely usual in scientific controversy Impartial opinion has certainly not endorsed all the sometimes extravagant claims made

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Prophylaxis —During an epidemic the avoidance of crowds especially indoors would seem to be advisable Children should be kept away from others as far as possible indeed the fewer people they encounter the better Food should be carefully supervised milk and water should be boiled and nothing eaten raw Public Health authorities do not advise closure of residential schools since while the individual may thereby be advantaged the general dissemination of infection which might follow could easily react adversely on the community Parents however often force their hands

The problem confronting a Public Health authority could not well be more difficult The fact is that we do not really know how the disease is spread and until we do our measures can hardly rise much above a series of pious gestures Until the electron microscope is a common item of equipment the detection of carriers (if carriers do play a vital part in spread) must remain a most elaborate and cumbersome procedure The strict disposal of excreta the destruction of flies and other insects the wearing of masks and so on are measures that will obviously do no harm and may do good But until the mechanism of dissemination has been finally and satisfactorily worked out it is obviously impossible to lay down any hard and fast

muscle of a group with common action is involved electrical stimulation may be included but voluntary exercises intelligently controlled form the basis of treatment which should be kept up for years Fatigue which retards recovery, should always be avoided Progress should preferably be recorded on muscle charts Only when no further recovery is likely should the patient be allowed up—which may mean confinement to bed for a year or even longer *It is important to note that a certain quantum of improvement can always be secured by treatment whether given im mediately or even after the lapse of years*

Diametrically opposed to this is the Kenny method which decrees that muscle pain is due not to inflamed posterior root ganglia but to spasm circulatory stasis and thus anoxia Fomentations or diathermy are applied to the painful muscles which are passively exercised from the outset in spite of pain When defervescence has occurred alternate hot and cold sprays in the bath are employed to restore circulation massage being avoided Passive and active move ments are practised from the outset carefully avoiding fatigue and at no time is the limb immobilized The patient is encouraged to place the feet to the ground and gradually to take the body weight as soon as possible Great emphasis is placed on helping the patient to overcome inhibition and to overcome the psychological barriers to recovery This method has its zealous supporters and equally determined opponents whatever its merits they have certainly been pressed by methods scarcely usual in scientific controversy Impartial opinion has certainly not endorsed all the sometimes extravagant claims made

appeared in the United States so as almost to merit the description pandemic But cases had appeared in England before then and have since continued to crop up though on markedly diminishing scale

Incidence—Any age but very rare in young children In the 1919 20 outbreak the second decade seemed to be much the most vulnerable period of life The sexes seem to be equally affected Infectivity is low and capricious

Pathology—The virus is said to gain access to the C N S by way of the axons of the peripheral nerves In the brain it is said to survive for very long periods and sometimes to show signs of renewed proliferation by clinical recrudescences or exacerbations Whether in such cases it can ever again emerge to infect other persons is uncertain Post mortem the grey matter of the cerebrum and sometimes of the cord shows a uniform rose pink injection the cerebral vessels are deeply congested and occasionally there are hæmorrhages Microscopically the most striking change is the perivascular cuffing —a ring of lymphocytes around each blood vessel The grey matter is invaded by microglia but there is no demyelination Sometimes the ganglion cells are destroyed The basal ganglia are usually heavily involved

At the height of the acute phase the cerebro spinal fluid is increased in pressure and amount and usually shows a pleocytosis Chemically the protein sugar and chlorides are within the limits of normal though the first two tend to be high Neither the pressure nor content of the fluid is of any prognostic value

Signs and Symptoms and Course—It must be

procedure All that we can do is to play safe take measures against either a droplet or ingestion infection and hope for the best

There remains the question of serum or vaccines Serum seems to have been generally abandoned as useless Vaccines consisting either of live attenuated virus or of killed virus have been prepared and have had variable success But since the disease only attacks a very small percentage of the population even in those countries where it is commonest the question of widespread vaccination has not so far been seriously considered though in a closed community where a small number only are at risk it might prove very valuable

Encephalitis Lethargica

An acute infection of the nervous system the brunt of which falls on the brain The acute phase is commonly followed by sequelæ after a lapse of months or years

Cause —A virus which when introduced to the nervous system of monkeys has reproduced the disease

Mode of Spread is assumed to be by droplet but the evidence is scanty and inconclusive Case-to-case infection is rare and carriers have been postulated as the agents of dissemination via their nasopharyngeal secretions

Epidemology —Epidemics have apparently occurred throughout history One which began presumably in Central Europe in 1917 spread slowly westward to reach this country in 1919 20 and subsequently

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Signs and Symptoms and Course —It must be

and indeed many of these patients arrive in hospital *by way of the juvenile or criminal courts*

Later on, whether behaviour disorder has intervened or not *post encephalitic Parkinsonism* develops Here it would appear as if the emotional hypersensitivity of the preceding stage were giving way to atrophy Cortical control over movement and muscle tone is steadily lessened so that muscle tone increases and the limbs and body flex and stiffen On attempting to extend the patient s arm the elbow joint seems as if it were working on a ratchet the arm straightening in a series of little jerks In a well marked case the appearance is typical The knees are bent and the arms flexed across and in front of the trunk the head is bowed on the chest and the body leans forward The patient instead of walking normally slides his feet forward in a series of rapid shuffles without lifting them from the ground He finds it difficult to start and once started to stop and in full career seems to be on the point of falling forward as if in pursuit of his centre of gravity Indeed he may only be able to bring himself to a halt by colliding with some fixed object The associated movements—swinging the arms in rhythm with the legs—are lost But it is the face which is most characteristic Whether in amusement or rage it is quite devoid of expression—Parkinson s mask—and it is sometimes a disconcerting experience to hear such a patient make a remark *indicative of an acute and witty intelligence which* not infrequently happens Speech is difficult and slurred (dysarthria) the voice is monotonous and saliva dribbles from the angles of the mouth The blinking reflex is lost and the whole countenance gives

an impression of unnatural and uncanny immobility As the condition progresses muscular action becomes more and more restricted and slow The tremor varies Usually it ceases on purposeful movement and during sleep but in normal inactive states it is incessant the first finger and thumb especially seeming to be continuously rolling pills In a certain percentage of cases these changes are confined to only one half of the body

A feature of both acute and chronic stages in some patients is the oculo gyric crisis Here the eyes may roll upwards or sideways and remain fixed in that position for ten minutes or for an hour or even longer In addition a large number of tics choreiform or athetoid movements worked into habit patterns may occur Mentally the disposition descends gradually to a uniform apathy broken by fits of acute depression attacks of weeping or outbursts of splenetic ill humour or rage Such patients in the mass tend to be exceedingly quarrelsome and often despite the most extreme disabilities come to blows among themselves This is evident even among cases which have apparently left the behaviour disorder stage far behind

Diagnosis —In the acute stage has to be made from other forms of encephalitis occurring in the virus infections from influenza from poliomyelitis and from meningitis of any kind The separation from other forms of encephalitis may be most difficult if not impossible in the absence of a history of the associated disease but this is usually readily forthcoming unless in such conditions as mumps where parotitis or orchitis have not preceded or followed the encephalitis From

influenza certain forms of the disease in which there are no nervous signs are apparently almost inseparable

In poliomyelitis the rapid development of flaccid paralysis and the alterations in the cerebro spinal fluid are characteristic doubt may more readily arise in abortive cases which however clear up rapidly Coccal meningitis should hardly confuse though tuberculous meningitis may present difficulty at first sight bacteriological and chemical examination of the fluid should soon however put the diagnosis beyond doubt

Prognosis—According to Von Economo of every ten cases seen in the acute stage four will die three will make a complete recovery and three will develop sequelæ Prognosis must therefore be guarded Sequelæ may follow even the mildest attack and some years must therefore elapse before the full extent of these are assessable Mild behaviour disorders are not incompatible with a reasonably satisfactory existence but severe disorders or the onset of Parkinsonism are of grave import The fact is that these patients of necessity become social outcasts in variable degree the former because they have to be kept under supervision and the latter because they gradually become so completely dependent on others and in the later stages physically and therefore socially repulsive Again while very mild cases may suffer little if any reduction in general health severe cases deteriorate steadily progressively lose their vitality and easily fall a prey to some intercurrent infection

Treatment and Management—During the acute phase this is primarily a matter of nursing and the patient is better isolated It is essential to see that he

takes sufficient nourishment and this may involve nasal feeding bed sores must also be averted so that an air or water bed or a sorbo mattress is desirable Repeated lumbar puncture sometimes seems to help but no drugs are of any avail

In the chronic Parkinsonian stage the rigidity which descends on the unhappy patient tends completely to immobilize him so that he may not be able to feed himself or even to swallow or speak The only drugs which influence the rigidity are stramonium or bella donna which have to be given in full doses One unfortunate result of this is that the saliva dries up the mouth becomes foul and the teeth decay Sus ceptibility to nasopharyngeal infections increases enormously and this coupled with the fact that such patients may show little febrile reaction to quite sharp infections may result in a patient developing a dangerous upper respiratory tract condition with few objective signs of it In general the temperature regulating mechanism of such patients is unreliable and easily deranged exposure to the sun may induce alarming pyrexia whereas as mentioned infection may meet with a trivial response

Prophylaxis —As mentioned the infectivity is low while the mode of transmission is uncertain On the assumption that a droplet mechanism is responsible for spread cases if recognized in the acute stage should be isolated and the attendants advised to wear masks though the risk of contracting the disease would seem negligible As it may yet prove to be an ingestion infection precautions against an enteral transmission should also be enforced

Note —Apart from lethargic encephalitis a number

of other forms of infectious (virus) encephalitis have recently been described especially in the U S A These show a different clinical picture in the acute stage, and sequelæ when they occur are also different from the foregoing The most common type, the equine does not occur in this country

SECTION X

RESPIRATORY INFECTIONS

Measles

MEASLES is an acute infectious disease characterized by sharp fever widespread catarrh of the upper respiratory tract and a typical rash

Cause—A virus which has been cultivated on chick embryo and with which the disease has been reproduced

Incidence—Mainly children from six months to five years Adults may acquire it but the disease is so widespread and infectious that few city dwellers fail to contract it in childhood One attack usually confers permanent immunity

Varieties—There are no clear cut distinctions to be drawn between cases of measles They may be *mild* or *malignant* In the latter death is said to occur before the rash appears and as usual hæmorrhages may occur from the mucous membranes One has never seen or heard of such a case In any event the term hæmorrhagic measles is usually if not strictly correctly reserved to rashes of exceptional heaviness into which hæmorrhages may occur and which consequently persist for a week or fortnight as hæmorrhagic staining These usually offer quite a good prognosis however To cases occurring in those almost completely protected by serum in which the catarrh

and/or rash are so poorly marked as to be equivocal the terms *morbilli sine catarrho* and *morbilli sine morbillis* may be applied A bullous form of measles has also been described (*morbilli bullosi*)

Incubation Period —From exposure to invasive catarrh ten to fourteen days from exposure to appearance of rash fourteen to eighteen days In *some cases, especially those partially protected by* serum these periods may be extended by a week

Segregation Period —Until the rash has faded or a week from its first appearance

Mode of Spread —Measles is most infectious *before the rash comes out* Although fomites may convey infection, spread is mostly by droplet Violent coughing and sneezing are a feature of the invasive stage hence the high infectivity

Pathology —The local lesion is situate in the naso pharynx whence the virus enters the blood Degkwitz by withdrawing blood on the first day of the rash and subculturing symbiotically with pneumococci (which he later filtered off) succeeded in infecting monkeys via the filtrate The skin and respiratory mucosa show a round celled infiltration The nature of Koplik s spots has not yet been satisfactorily deter mined but they are believed to represent the inspis sated secretion of tiny buccal glands The blood shows a leucopenia affecting especially the poly nuclears with a relative lymphocytosis The plasma cells are also increased The Wassermann reaction is often temporarily positive

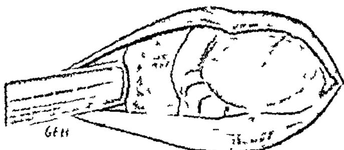
The pneumonia of measles is usually due to the Pfeiffer bacillus the hæmolytic streptococcus or the pneumococcus any of which may be quick to exploit

the breach in the defences made by the virus When the Pfeiffer organism is concerned a focal interstitial pneumonia develops in which the alveoli may be clear but so compressed by interstitial proliferation of fibroblasts and endothelial cells as to be almost obliterated Similar aggregations about the bronchi may so compress the latter as to produce areas of collapse Clinically such cases are marked by cyanosis out of proportion to the lung signs Where pneumococci are active the familiar lobar consolidation may ensue Where streptococci are the dominant organisms there is patchy congestion about certain bronchioles the alveoli of which may be full of pus Areas of consolidation compensatory emphysema and collapse may succeed one another throughout the lung Most lung abscesses and empyemata in measles are due to streptococci whether these have been the first invaders to follow the virus or whether they intervene later on In epidemics any one of the above may be the predominant secondary invader thus colouring the clinical picture appreciably

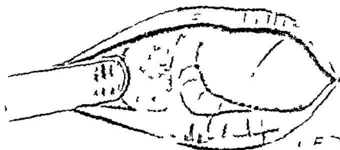
The pathological changes in a fatal case will depend on which complication was responsible for death This may be a variety of pneumonia an otitis media and mastoiditis which has led to meningitis or a cerebral abscess or perhaps an enteritis In the last case the changes will be those detailed on page 202 The blood shows a polynucleosis with increase of the large lymphocytes and a marked diminution in the small lymphocytes (in uncomplicated cases)

Course —(a) *Stage of Invasion* —The temperature rises steeply and a generalized catarrh sets in which

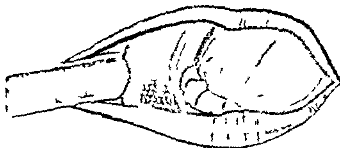
spreads rapidly throughout the whole respiratory tract from the conjunctivæ to the terminal bronchioles, and so it is said through the whole digestive tract as well Hence there is *conjunctivitis* with blepharitis coryza with *sneezing* and a short dry *cough* Should the larynx be severely congested this cough may rapidly become croupy An invasive *enteritis is not uncommon Very soon the enanthem is* well established The tongue is inflamed and thickly furred the mucous membrane of the entire oropharynx is a bright angry red, sometimes showing on the cheeks patches of deeper injection resembling the exanthem yet to develop In about twenty four hours *Koplik s spots* appear (see Plate XIV A and B) These at first scanty show up as tiny white flecks like grains of coarse salt against the granular inflamed background As originally described by Koplik each spot is surrounded by a ring of injection and shows a bluish white point in the centre but to most observers the spots appear a dirty white, and even occasionally a grey Sometimes they are aggregated in coarse flecks like aphthæ which however cannot be removed with the spatula They are best seen by reflected daylight and usually persist till the true rash appears and for a variable period thereafter Coincident with the Koplik s spots a *prodromal rash* may develop This is most commonly scarlatiniform though with a decided brownish tinge very rarely it may be macular mimicking the rash proper yet to appear exceptionally it may be urticarial It may fade quite rapidly or persist until evicted by the true rash The stage of invasion in general may last for three or even for six days the pyrexia pulse rate and respirations



A



B



C

PLATE XIV

A. Koplik Spots—Mu[illegible] gr[illegible]an [illegible] Spot [illegible] th[illegible] co[illegible]rse
B. An[illegible] t[illegible] typ[illegible] [illegible]f Koplik's Spots—[illegible] the buccal mucosa [illegible] [illegible]ety and the [illegible]pots are [illegible]ars
C. [illegible]llow points on [illegible] [illegible]al muc[illegible] m[illegible] be m[illegible] taken f[illegible] Kopliks

F[illegible]

steadily increasing so that if the catarrh is slight unless a prodromal rash appears or the physician has observed the enanthem he may mistake the case for one of pneumonia The invasive stage is terminated by the eruption of the true rash Fine sparse discrete brick red macules appear behind the ears and along the hairline with a few isolated lesions irregularly scattered over the face neck and upper chest Very rapidly further macules appear enlarge and coalesce with their precursors to form blotchy irregular patches over the face and trunk while remaining mostly discrete on the limbs Indeed in a heavy rash only a few islands of unaffected skin may remain other than on the distal limbs The lesions become distinctly maculopapular and cover the entire body

The appearance in this stage (of advance) is very characteristic (see Plate XV) The face is puffy and covered with a heavy blotchy rash the eyelids are swollen the palpebral fissure is narrowed and the eyes appear sunken The conjunctivæ are injected and the lashes are often matted together with thick mucopurulent discharge There is often a streaming rhinorrhœa The lips are dry and the respirations hurried The unhappy child looks and feels utterly miserable he avoids the light and bitterly resents examination A typical short barking cough occurring singly or in little runs of two or three may often suggest whooping cough Note that if hæmolytic streptococci are proliferating in the oropharynx, the tongue may simulate that of scarlet fever

As the rash appears the general condition may remain stationary or deteriorate (The statement sometimes made that it automatically improves is

without foundation in one's own experience) Usually in the absence of complications it remains stationary for a few days until the eruption begins to fade When this occurs the fever abates, the temperature falls by lysis and the appetite returns In a further two or three days nothing but a brown 'staining' is left and the general condition has greatly improved *Indeed the rapidity with which recovery is made once the rash fades is most striking* Within a fortnight of the initial exanthem the child may be as well as ever, though even then especially in the so called hæmorrhagic cases staining may still persist

Complications—**Blepharitis, conjunctivitis rhinitis stomatitis,** and **bronchitis** are so much a part of all but the very mildest cases that it is hardly correct to call them complications They vary greatly in severity especially the conjunctivitis which if sharp may be followed by **corneal ulcer** often of a destructive type The most serious complication is undoubtedly pneumonia—usually a **bronchopneumonia** In practice this can usually be diagnosed only when, the rash having faded the polypnœa and fever persist because examination of the chest may furnish very little information for several days Occasionally moist crepitations or a patch of dullness with tubular breathing *may be found while the rash is till bright* but more commonly there are only a few dry rales or merely 'harsh breath sounds'—signs which may mean little or much The wise physician will therefore not commit himself until the rash has faded since even cases with urgent symptoms may then rapidly improve

Empyema is not an uncommon sequel to the fore

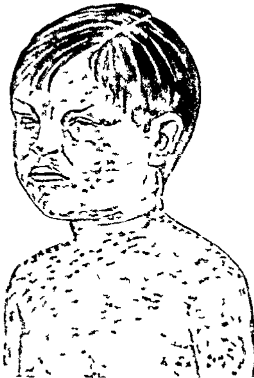


PLATE XX

[illegible]

F g p 80

going especially in streptococcal infections **Otitis media** is frequent often followed by **mastoiditis** **Laryngitis** may occur either before the rash due to congestion or after the rash due to diphtheria or ulceration The following aphorism is of value —

croup before the rash is *invariably measles croup after* the rash is diphtheria croup *with* the rash may be either This, if not invariably accurate draws attention to the frequency with which diphtheria may be engrafted on to the devitalized larynx of measles to form a very dangerous combination **Enteritis** is another disturbing complication which often intervenes *in bronchopneumonia to render a hitherto* favourable prognosis hopeless A demyelinating **encephalitis** is an uncommon and late event prone to show a high mortality **Cancrum oris** is a time honoured sequela now rarely (if ever) seen due presumably to the infection of a dental or other ulcer with the twin organisms of Vincent s Angina (q v) The liability of diphtheria to supervene has been noted another sinister combination is that of measles and whooping cough The lighting up of a pulmonary tuberculous focus sometimes spoken of as frequent *is a rare but fatal event since the infection is prone* to generalize **Fibrosis** of the lung is a common sequela and the development of subsequent **bronchiectasis** is by no means rare

Attenuated measles is seen in those who have had a dose of convalescent measles serum or some other prophylactic which was either too small in amount or given too late to secure complete protection Here all that may be seen is a very slight catarrh a very sparse and fleeting rash and perhaps only a few

Koplik's spots—or none at all In diagnosis the history of exposure and of the prophylactic injection is all important Complications in these cases are very rare but it should be noted that they can give rise to unmodified measles in other children and therefore must be dealt with as if they were suffering from an ordinary attack

Diagnosis —The diagnosis of measles should only be made if in addition to a typical rash respiratory tract catarrh is or has been present The conjunctivitis and blepharitis have already been mentioned The presence or history of sneezing and coughing is most important measles without them is almost unthinkable The cough furthermore is distinctive The student must familiarize himself with it and also with *Koplik s spots* These last are absolutely diagnostic Rarely he may be deceived by food debris thrush aphthæ or small ulcers and very rarely by yellowish points of unknown significance which occur far back on the buccal mucosa (see Plate XIV) If he will remember the following points he will seldom go wrong (*a*) Koplik s are *fine* spots about the size of a grain of salt even if aggregated in coarse patches there are always some fine ones to be seen (*b*) they cannot be removed with a spatula as can food debris (*c*) they occur not in a normal quiet mouth but on an angry inflamed buccal mucosa often granular or velvety (*d*) they are most numerous about the middle of the cheek or in the folds at the bases of the gums and may (rarely) be seen on the conjunctiva and (*e*) they are grey or white very rarely yellow In any event if the student misdiagnoses a case as measles, relatively little harm is done *it is the reverse*

which is so common and so often disastrous Measles is a dangerous disease superadded to a severe existing illness it may easily lead to disaster *The student should form the habit of looking for Koplik s in every child with a rash* At epidemic times every sick child should be suspected indeed there is no disease which more consistently dupes the clinician Measles may masquerade as —

(1) *Laryngeal Diphtheria* —Every croup seen during an epidemic should be rigorously scrutinized for Koplik s spots

(2) *Scarlet Fever* —The prodromal rash and the tongue may mimic scarlet fever to perfection though to the experienced eye the rash often tends to have a salmon pink or brownish tinge

(3) *Whooping Cough* —The invasive cough often tends to occur in little spasmodic runs extremely suggestive of whooping cough

(4) *Rubella* —Attenuated measles especially in verminous children with resultant chronic suboccipital and posterior cervical lymphadenopathy may be almost impossible to differentiate from rubella Here the history of prophylactic serum is all important Remember also the age group Rubella is rare in children under five years (see p 299)

(5) *Drug* rashes and *serum* rashes are often simulated

The foregoing are errors frequently committed and not always by the totally inexperienced They can all be avoided by thinking of measles and looking for Koplik s spots The occasions when these will be absent unless in the earliest stages of attenuated cases are negligible Even then the buccal mucosa

may show a suspicious granularity or a lack of lustre that means much to the experienced eye

Prognosis—Most deaths from measles occur in children under two years though up to five the risk is appreciable The outlook is fair in uncomplicated bronchopneumonia but very bad if enteritis intervenes Secondary laryngitis is also of bad omen

Treatment—Is that of the febrile state The toilet of the mouth and especially the eyes should be instituted at once—frequent bathing of the latter with the application of Ung Hyd Ox Flav dil being of much value Since most bronchopneumonia is the result of secondary coccal invasion penicillin or sulphonamide derivatives may be given prophylactically Should bronchopneumonia develop the case is better isolated as amongst the debilitated children in a ward it may display a decidedly infectious character Cases with enteritis and purulent discharges should also be segregated Laryngitis may require operative measures coming on after the rash it is best regarded and treated as due to diphtheria

Prophylaxis—The blood of those who have recovered from measles contains antibody during life the more recent the attack the greater the antibody content The blood of recent convalescents or adult immunes is therefore drawn off and the serum is separated A Wassermann is carried out and if positive is repeated on the patient a month later when it is usually negative The serum can then be desiccated and stored An extract of fresh human placenta (immune globulin') contains similar antibodies in the same proportion as adult serum i e about half

that of recent convalescent serum On the other hand Gamma globulin may contain double Any one of these if sufficient be injected within the first five days from exposure will usually prevent measles developing and even within the next two or three days will attenuate an attack Later than this they have no effect * and are entirely valueless in treatment once the disease has set in The dosage is roughly 5 c c of convalescent serum or 10 c c of adult serum for chilren under three years Smaller doses will attenuate but not prevent Older children require 2 and 4 c c respectively of serum or placental extract per year of age No child under five should be allowed to contract unmodified measles though if exposed it is probably best to allow it to experience an attenu ated attack and thus acquire some immunity No child under two should be allowed to contract measles at all since the mortality at that age is so high The same applies to patients of any age seriously ill with any other disease It must not be forgotten that in the absence of a suitable serum or extract parental whole blood can be injected intramuscularly in the propor tion (roughly) of 20 c c whole blood to 10 c c of adult or 5 c c of convalescent serum

Attempts have also been made to produce a suitable vaccine so far with some but not conspicuous success Once again it is a question of striking a balance between a preparation so virulent as to set up an attack of the disease and a preparation so attenuated as to confer virtually no immunity No doubt in time a satisfactory compromise will be evolved

Control of Outbreaks —Segregate the offender

* Unless intravenously in hero c doses

Quarantine for three weeks The ideal is completely to protect all those children under two years or seriously ill, and partially to protect the remainder, segregating all *individually* Usually this is impossible in which case both groups as a whole should be separated Unfortunately only some of the partially protected group will usually develop attacks thus re exposing the remainder whose passive immunity will by then be on the wane so that they may develop severe unmodified measles unless they are again partially protected Two cautions are necessary —(1) *never accept a history of measles* unless from an unimpeachable source—certainly not from the mother A history of having been in hospital with measles is not enough—enquire from the hospital, (2) when serum has been given examine every child carefully from the tenth day onwards It is very easy to miss an attenuated attack Remember also that the incubation period may be greatly prolonged—one has seen it extend to twenty five days in one instance

Debré s Phenomenon —If 0 2 c c of convalescent serum be injected intradermally into a child in the catarrhal stage the rash when it appears will avoid that area

WHOOPING COUGH

Whooping Cough (pertussis) is an acute infectious disease primarily affecting the respiratory system though the nervous system is also involved in greater or lesser degree Commonly regarded with com placency, if not with contempt, the disease is very

fatal to infants and small children The death rate per 100 cases during the years 1932–1938 in the L C C hospitals was more than twice that of measles or diphtheria and nearly twenty times that of scarlet fever

Cause —Whooping cough has all the stigmata of a virus disease Thus it is extremely infectious via droplets in the early stages but very much less so later on it is characterized by a relative lymphocytosis one attack confers lifelong immunity carriers are unknown it has a long incubation period convalescent serum is of value in prophylaxis but useless in treatment and an encephalitis occasionally follows it Nevertheless there is considerable evidence that it is due to a small cocco bacillus the *Hæmophilus pertussis* (bacillus of Bordet Gengou) which curiously enough is a first cousin of the bacillus bronchi septicus for long believed to be the cause of canine distemper and of the Pfeiffer bacillus similarly held accountable for influenza Since both these conditions have since been shown to be due to viruses it is tempting to believe that family history may repeat itself On the other hand the *H pertussis* would appear to be recoverable from the droplets in almost every case of whooping cough especially during the catarrhal period after which it gradually disappears until after the sixth week it is rarely to be found This corresponds roughly with infectivity as clinically observed Again many observers have claimed to have produced whooping cough in apes by the intra tracheal instillation of cultures of *H pertussis* and finally a worker in the United States has performed what may be a conclusive experiment on his four

children He first of all immunized two of them with vaccines made from *H pertussis* and subsequently instilled fresh cultures of *H pertussis* intranasally in all four as a result both 'unprotected' children developed whooping cough whereas the protected children did not It is, of course possible that a virus living symbiotically with the *H pertussis* may still be the primary cause though complement fixation and agglutination tests with the latter are available in the third week of the disease It would seem in fact that the *H pertussis* all but fulfils Koch s postulates but not quite Certainly the vaccines hitherto available in this country have not reproduced the success claimed for those produced in the United States but the matter is still under investigation

Pathology and Morbid Anatomy—There is little that is distinctive There is moderate respiratory catarrh with areas of collapse and of compensatory emphysema Radiologically the bronchial glands appear enlarged (though they are often normal at post mortem) the hilar shadows are thickened and striated and a developing bronchiectasis is not uncommon in convalescence following bronchopneumonia This is not surprising since an interstitial bronchopneumonia similar to that often encountered in measles and influenza (due to the closely related Pfeiffer s bacillus) is a frequent complication and in such a pneumonia the bronchioles may readily weaken and degenerate Cases dying of convulsions merely show congestion of the cerebrum as a rule though a multitude of pathological changes has from time to time been described The convulsions so characteristic of the disease have been attributed to (1) anoxaemia,

(2) cerebral hæmorrhage (3) spasmophilia—deficiency of Ca and P in the blood (4) a meningo encephalitis and (5) spasm of the cerebral blood vessels especially in conjunction with (6) degeneration of the cornu ammonis They might also be due (7) to alkalosis consequent on vomiting but the theory of the moment is that they are due to (8) the endotoxin of the *H pertussis* Since this organism lies on and does not penetrate the mucosa it is difficult to see how the endotoxin is absorbed to any great extent (There is no exotoxin produced)

Quite apart from convulsions a virus type demyelinating encephalitis has been described (surely an unheard of event in non virus infections) The *blood* may show up to 16 000 white cells of which 60 per cent are small lymphocytes

Incubation Period —Very indefinite Said to be seven to fourteen days

Segregation Period —A month from the first whoop or six weeks in all is usually safe Alternatively until a cough plate is negative

Incidence —Any age but mostly children in the first three years of life

Mode of Spread —Mainly by droplet especially during the preliminary catarrhal stage

Course —This resolves itself into two stages In (*a*) the **catarrhal stage** the child develops a simple *cough* which is noted to be *worse at night* and is often followed by *vomiting* There may be slight irregular pyrexia occasionally an invasive laryngitis occurs As time goes on the cough tends to occur in runs which become progressively longer and in about a fortnight the (*b*) **paroxysmal stage** develops The

paroxysms are typical The child seems to know when one is coming on, he often struggles to a sitting posture grasps the cot side for support and usually hangs his head He then takes a preliminary breath followed at once by a diminishing series of short explosive uncontrollable coughs, during which the face becomes increasingly more livid and congested the tongue protrudes the eyes bulge and the tears roll down the cheeks Following the last and smallest cough there may be a pause of a second s duration, which is terminated abruptly by the whoop—a high pitched crowing inspiration through a spasmodically contracted glottis Usually several paroxysms follow one another in series until the child succeeds in bringing up a pellet of thick tenacious mucopus which he may hardly have the strength to expectorate The pellet, though small is sometimes as large as the child s trachea and the paroxysm often terminates in vomiting and incontinence after which the child falls back exhausted though only for the moment He usually recovers surprisingly rapidly and may appear quite normal between the bouts But since as many as fifty paroxysms may occur in a day, his nutrition if vomiting is a prominent feature may suffer seriously though fortunately nothing like so much as it would were the condition febrile Marasmus may however sometimes develop Fear excitement a *spatula in the mouth or the* example of others may initiate a paroxysm

In severe cases with numerous paroxysms, the appearance is characteristic the face becoming puffy and slightly cyanosed the lips thick the eyes heavy the eyelids puffy and the expression dull The attacks

occur daily with increasing frequency up to a maximum which may be reached in three or four weeks thereafter they gradually diminish rarely they cease abruptly The whole course usually covers about two months but it is important to remember that for as much as perhaps a year after recovery the child may (possibly not always unintentionally) astonish his elders by exhibiting a typical paroxysm especially if ill thwarted or annoyed On examination nothing at all may be discovered in the chest Alternatively loud harsh rales may be heard Areas of collapse are by no means uncommon especially over the lower lobes they vary from day to day and are doubtless due to blockage of a large bronchiole since following a bout of coughing the air entry may become normal A chest picture which changes from day to day is typical of pertussis A common finding is **acute cardiac dilatation** especially during a severe paroxysm which may sometimes account for the vomiting Heart failure however unless as part of circulatory failure in bronchopneumonia is exceptional

Varieties —*Pertussis infection may not infrequently exist with a cough which remains simple throughout* In other instances the cough may be spasmodic but no whoop develops Babies sometimes experience attacks of paroxysmal sneezing without a cough This is exceedingly rare

Complications —These may be divided into (a) toxic complications possibly due to a neurotoxin elaborated by the organism or should the agent prove to be a virus to its invasion of the central nervous system (b) *infective* lesions produced by secondary invaders and (c) *mechanical* results following the

violent alterations in pressure in various parts of the body as a result of the spasms

"**Toxic**" complications consist chiefly of **convulsions**. These are especially liable to occur in the younger patients, especially under one year, when a 'fit' may replace a paroxysm. They occur usually but not invariably, in children with frequent and severe paroxysms and may rarely be the first indication that the paroxysmal stage has supervened. Unlike the paroxysms, convulsions are especially common in pneumonia. They must always be regarded with anxiety: the patient may easily pass into a convulsion from which he is only released by death.

Infective complications are common. As previously noted, an interstitial **bronchopneumonia** due to the *H. pertussis* is frequent; this may be intensified or overshadowed by the invasion of hæmolytic streptococci producing suppurative lesions such as **lung abscess** or **empyema**. When pneumonia develops the paroxysms of coughing and whooping cease, but reappear on recovery. As in measles, an intractable (and presumably infectious) **gastro-enteritis** may develop; *this alters the prognosis immediately for the worse*. **Marasmus** may in consequence ensue, so that even if the pneumonia should abate, the prognosis remains gloomy.

Complications resulting from the *physical* effects of the paroxysms are many. During a bout of coughing the pressure in the abdomen, thorax, head and neck may rise steeply. **Hernia**, umbilical, femoral or inguinal, or **prolapse** of the rectum may ensue. **Pulmonary emphysema** in some degree is common,

but surgical emphysema, like spontaneous pneumothorax is rare

Hæmorrhages are not infrequent They may occur from the nose or ear or from the lung A subconjunctival hæmorrhage may cause unfounded alarm it usually clears up without incident but a retinal or (in the elderly) a cerebral hæmorrhage is obviously of graver significance From chafing of the protruded tongue against the lower teeth during a paroxysm a **sublingual** (frænal) **ulcer** commonly ensues

Sequelæ —As noted **bronchiectasis** is a frequent occurrence in convalescence indeed some observers believe that most adult bronchiectasis derives from pertussis in childhood A tuberculous focus may light up to produce active generalized tuberculosis but whether whooping cough predisposes to subsequent pulmonary tuberculosis or not is uncertain Marasmus may ensue whether bronchopneumonia is present or not Children suffering from or convalescent from pertussis contract measles very readily the association is usually very unfavourable

Prognosis —The most important single prognostic factor is *age* Fully half the deaths occur in children under one year who must consequently be regarded as bad risks After the age of three the prognosis improves considerably The onset of convulsions is a sinister event more than half the cases in which convulsions *recur* come to a fatal issue Bronchopneumonia especially if complicated by enteritis is ominous even enteritis alone is highly dangerous

Diagnosis is simple once the paroxysmal stage has developed the whoop being unmistakable If as

sometimes happens the whoop is absent, the spasmodic cough perhaps followed by vomiting is readily recognized a spatula placed far back on the tongue will usually elicit a spasm Unfortunately diagnosis when most desirable is most difficult—i e in the catarrhal pre spasmodic stage when infectivity is highest Then the patient often coughs only at night a *cough* therefore that *persists* that is harsher than the signs (if any) in the lungs warrant that produces congestion of the face and that is *worse at night* must arouse grave suspicion If it is *followed by vomiting* suspicion amounts almost to certainty Conditions which may produce suggestive coughs are (*a*) measles —when Koplik s spots are usually present and (*b*) pressure on the trachea by enlarged glands this last is very rare Laryngismus stridulus ought not to confuse

Laboratory aids are three in number **Cough plates** are shallow circular glass dishes containing Bordet Gengou medium which must be made with *fresh* defibrinated horse or rabbit blood They are held in front of the child s mouth as he coughs and rotated slowly to secure even distribution of the droplets (The terminal pellet of sputum usually contaminated with other organisms must be avoided) The plate is then incubated In the early stages an almost pure culture of *Hæmophilus pertussis* should be obtained in about three days

Blood Count —This shows typically a high white count with 60 per cent or more of lymphocytes Unfortunately this is not constant until the catarrhal stage is over and so is useless in early diagnosis Note also that the lymphocytes may normally be as high as 50 per cent in young children

Complement fixation tests are positive from the third week onwards Here again while useful in confirmation they give no help when help is most required—in the catarrhal stage

Treatment—This must be on general lines The child is best kept in bed until the severe and frequent paroxysms are over An abdominal binder may be applied *An attendant should lift the child to a sitting posture immediately a paroxysm begins* otherwise he may vomit and inhale the vomit with probably fatal results He should be trained to expectorate into a receiver the mucus should be wiped away and the face cleaned and dried when the paroxysm is over Should vomiting be frequent the maintenance of the child s nutrition often becomes a matter of anxiety Washing out the stomach has been recommended but gives uncertain results Feeds should be small fluid or semi fluid frequent and follow vomiting in the hope that they will pass through the stomach quickly and escape expulsion at the next bout of coughing Vitamin concentrates have been strongly recommended and should be included

Many and numerous are the drugs which have been vaunted as controlling the spasms Probably they are all useless such at any rate has been one s own experience Large doses of luminal may have some effect but it is to say the least not striking Some sort of expectorant is usually exhibited whatever its effect on the paroxysms it may very easily set up a gastritis in a young child which is no negligible matter in this disease The physician may consider himself fortunate if he can prevent pneumonia or

enteritis developing Open air nursing when feasible, is a definitely helpful measure and sulphonamides may well be given prophylactically should pneumonia appear to be developing Here also the oxygen tent may be of considerable value Enteritis should be ruthlessly segregated In a convulsion the old fashioned hot mustard bath may be of great value and intramuscular luminal may be administered Lumbar puncture *may* arrest a convulsion but is not without its dangers

Radiation of the lung bases with the X rays has been claimed by some to give exceptionally good results Ultra violet ray therapy may be of benefit in the later stages but should not be given in the early stages at any rate Convalescent serum and vaccines have also been recommended in the early stages but have not secured general approbation The truth is that the multiplicity of remedies advanced and the spate of new ones which continually appear (including the much publicized flight in an aeroplane) is the best proof that none is of striking efficacy

Prophylaxis is more hopeful though the skin tests for susceptibility analogous to those for scarlet fever and diphtheria have hitherto given equivocal results In prophylaxis several types of vaccines have been employed but reputedly the most successful (Sauer s) is made from Phase I organisms grown on Bordet Gengou medium enriched with human instead of horse blood This has been the subject of enthusiastic reports in the U S A where in some areas it is stand ard practice to employ it for the immunization of children of pre school age for which purpose some local authorities supply it free Vaccines made in an

identical manner in this country have not given comparable results but further trials are being made Convalescent serum when available is of undoubted value and hyperimmune serum made by immunizing persons who have had the disease with vaccines so as to push their immunity still higher has achieved an excellent reputation Unfortunately pertussis is normally so capricious in its infectivity (so far as frank clinical attacks are concerned at any rate) that the evaluation of any given prophylactic measure is extremely difficult

Control of Outbreaks—The offender should be segregated Impose a quarantine on the contacts of sixteen days Any child who develops a cough should be isolated *at once* for observation Give convalescent serum if available prophylactically

Note on Coughs

There are certain coughs characteristic of certain conditions with which the student would do well to familiarize himself They are (1) the simple cough of tracheitis as in the common cold (2) the short bark of measles (3) the *croupy* cough of laryngeal diphtheria measles or catarrhal laryngitis (4) the *spasmodic* cough of whooping cough (5) the spluttering cough of pharyngeal paralysis (6) the short suppressed cough of pneumonia with pleurisy (7) the deep loose liquid cough of cavity formation in pulmonary tuberculosis or bronchiectasis (8) the wheezy semi spasmodic cough of chronic bronchitis (9) the gander cough of pressure on the trachea and

(10) the "bovine" cough of recurrent laryngeal nerve palsy The student should always note whether the cough is productive of sputum or not and if so the amount character and consistency of the sputum produced The presence or absence of blood should also be established Coughs can be very informative

SECTION XI

RUBELLA GLANDULAR FEVER AND MUMPS

RUBELLA

RUBELLA (German measles) is a mild acute infection characterized by moderate pyrexia a rash and enlargement of the superficial lymph nodes

Cause —A virus which it has been claimed has been successfully grown on chick embryo the culture subsequently reproducing the disease in monkeys One attack protects for lif

Incidence —Older children and young adults as a rule Epidemics are common usually in spring

Incubation Period —Fourteen to twenty one days usually seventeen

Segregation Period —Until the rash has faded A week is ample

Mode of Spread —Droplets and direct contact The disease varies greatly in infectivity which is at its highest just before the rash appears

Pathology —Little is known The nasopharynx is the site of the local lesion whence the virus probably enters the blood This shows a leucopenia with a relative lymphocytosis and a pronounced increase in the plasma cells The W R is often positive

Course —The stage of invasion is short and often unperceived Adults however may sometimes be febrile for some days and complain of a stiff neck (due

to the adenopathy) which they usually attribute to a ' cold or ' flu ' There is often coryza and a " dry throat which may occasionally prove severe should secondary invaders be present The *rash* appears first on the face and neck along the hair line thence it spreads over the face generally the trunk and lastly the limbs The circumoral area is invaded Classically the rash consists of fine discrete salmon pink macules intermediate in size between the coarsest punctations of scarlet fever and the macules of measles (see Plate XVI) The colour likewise lies midway between the two These spots may run together to form lines ' and blotches ' though never to the same extent as in measles But it is dangerous to be dogmatic about the rash in rubella for a prime characteristic of the eruption is its impermanence and its liability to mutation Thus on the first day typical fine macules are to be seen on the face and trunk and sparsely or the limbs On the second day the face may be clear of the rash which has now become confluent on the trunk and limbs closely simulating scarlet fever On the third day there may be nothing at all to be seen Hence the aphorism— first day measles second day scarlet and the third day gone as a summary of the rubella rash

The *adenopathy* usually precedes or accompanies the eruption The glands affected are principally the suboccipital group at the base of the skull and the posterior cervical group in the posterior triangle of the neck Often diagnostic is enlargement of the small lymph node over the mastoid process The axillary and inguinal nodes less often share in the adenopathy The enlarged glands remain discrete



PLATE XVI

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and fairly soft the enlargement is very moderate as a rule occasionally they may be seen over the mastoids but usually they can only be felt Thus the posterior cervical chain can often be felt like a string of beads The glands are tender and in adults especially this tenderness may persist for as long as a week after the rash has faded Suppuration does not occur The last of the triad of physical signs is an expression of the mild catarrh typical of rubella—the pink eye This consists of a salmon pink injection of the angular conjunctiva In contrast with *measles* there is neither lachrymation nor discharge the eyes are quiet and the lids are normal

Moderate pyrexia often accompanies the invasion to abate when the rash appears or certainly when it fades But often in children pyrexia is absent Even if it is high the patient is not febrile or toxic he looks and feels well wants to get up and has a good appetite Very occasionally an adult (possibly because of the proliferation of secondary organisms) may develop a sharp angina and high fever Again very occasionally the rash may remain for as long as a week as a macular erythema and staining may ensue Some fine desquamation on the trunk is usual again occasionally it may be profuse and pinhole on the palms and soles though this is exceptional

Complications with one exception are usually rare and trivial Slight **bronchitis** may occur **Arthritis** is rare So too is a demyelinating **encephalitis** A simple **purpura** may also occur

The exception refers to the pregnant woman Should she develop rubella during the first three months and more particularly during the first eight

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weeks of pregnancy disasters to the growing embryo may readily ensue These may consist, so it is reported, of congenital defects of any kind but the commonest would appear to be developmental anomalies of the heart congenital cataracts and other eye defects, and even deaf mutism Apparently the virus interferes very seriously with the nutrition of the growing cells but its mode of action has not so far been finally worked out since it is only within the past few years that its malignant rôle in this respect has attracted attention

Diagnosis —In typical examples this is easy otherwise it may be most difficult There is no doubt that rubella is frequently misdiagnosed Transient macular rashes arise from many causes known and unknown The value of the adenopathy as a diagnostic sign is vitiated by the fact that firstly in children the lymph nodes are normally larger and more prominent than in adults and secondly in verminous children the suboccipital posterior cervical and inguinal glands are often chronically enlarged and palpable The mastoid gland when it is affected is pretty well conclusive This may be enlarged as a result of adjacent sepsis and sometimes in glandular fever (q v) In the latter the gland is visibly swollen there is (usually) much more fever of much longer duration and the rash when one is present does not follow the typical development of the rubella rash The commonest single cause of confusion is measles but here the catarrh blepharitis Koplik s spots and toxæmia should easily allow of differentiation unless in very attenuated cases with little rash or catarrh when sometimes it may be frankly impossible (in the

absence of a *history of serum)* The diazo reaction usually present in measles but *absent in rubella* will help as will a blood count showing in measles a moderate but in rubella a pronounced increase in plasma cells The count should similarly exclude doubtful *food* drug or serum rashes which show an eosinophilia to *supplement the history* but which may otherwise prove very confusing *The glands* will not of course be enlarged Scarlet fever may also be closely simulated by the second day rash of rubella *especially if* streptococci are active when the tongue may abet *the deception* Here the negative Schultz Charlton the failure (on *observation) of the Dick* test to swing from positive to negative the adenopathy and the plasmacytosis should differentiate rubella Desquamation be it noted is not to be relied on Most *difficult at first* sight may be the roseola of syphilis which with the adenopathy may mimic rubella most convincingly The *persistence of the* syphilitic rash will of itself arouse suspicion after the *first few* days Further in rubella the eruption may itch and the glands are tender An immediate diagnosis may turn on the state *of the* mastoid and epitrochlear glands implication of the former *favouring* rubella and of the latter syphilis Of the other signs *of lues a* positive Wassermann unless persistent is not conclusive Cases of *leukæmia* which develop a rash should be differentiated by the *great increase* in white cells shown by the blood count and the *enlarged* spleens usual in these conditions though sometimes immediate diagnosis may be impossible Pityriasis rosea which shows a *herald* patch and a much coarser eruption should not confuse

Prognosis —Invariably good

Treatment —Rest in bed on light diet for as long as pyrexia persists is all that is required

Prophylaxis —Convalescent serum is of value Hitherto it has hardly been considered worth the trouble but in view of the vicious effects of rubella on the developing embryo no doubt serum will in future be stored for the protection of prospective mothers against the disease

Control of Outbreaks —Prompt isolation of sufferers and quarantine of contacts for three weeks should suffice Bed isolation should prevent spread in a ward Children may attend school for the first twelve days from exposure

Glandular Fever

Glandular fever (infectious mononucleosis) is an acute infectious disease characterized by fever a generalized enlargement of the lymphatic glands and a large increase in the mononuclears of the blood It belongs to a group of infections including rubella and such conditions as the recently described acute infectious lymphocytosis which are marked by changes in the non granular white cells of the blood and sometimes by changes in the serological reactions In glandular fever the lymph nodes are much more enlarged than in rubella while a rash is occasional only whereas as rubella it is invariable In both conditions there is a mononucleosis but whereas the small lymphocytes predominate in rubella the large lymphocytes are most prominent in glandular

fever As between the glandular fever cells and the plasma cells of rubella it takes an expert to differentiate Finally in both the Wassermann may be weakly positive From the wide clinical divergences between the various types of glandular fever described it seems likely that we are dealing with infections by a group of related viruses which require much additional elucidation

Cause —So far unknown but everything points to a virus As in other virus diseases notably mumps the clinical picture may vary according to the system or organ attacked Abortive cases also occur and appear to be common in epidemics

Incidence —The disease attacks children and young adults males preponderating It occurs sporadically but also in small epidemics especially in closed communities In such epidemics many apparently unaffected persons show blood changes—serological or cytological

Incubation Period —Five to ten days

Pathology —The glands are enlarged but discrete and leathery Histologically the germ centres are replaced by mononuclears some of them showing mitosis (There is never any suggestion of suppuration) The *blood* shows at first a moderate leucocytosis with all the elements equally increased The characteristic picture which later develops shows a leucocytosis up to as much as 20 000 per c mm with a relatively considerable reduction of neutrophiles and a great increase in the mononuclear cells The typical glandular fever cells are as large as monocytes the nucleus is immature and the cytoplasm is strongly basophil There are also halo cells—large lymphocytes in

which the cytoplasm stains only at the edges leaving a halo around the nucleus Mononuclears may comprise 70 per cent of the total leucocytes The red count is normal Sometimes there is a leucopenia

Signs, Symptoms and Course —The onset is usually sudden with fever and a generalized catarrh of the upper respiratory tract but especially of the fauces and pharynx so that sore throat is a prominent sympton The fever is sharp—there may even be rigors—as well as severe headache and pains in the limbs The catarrh may extend to the eyes producing conjunctivitis After some days the upper cervical lymph glands begin to enlarge to be followed by the lower cervical the axillary and later on the inguinal The submaxillaries and suboccipitals may also participate in the enlargement which is considerable sometimes to the size of a small egg The glands are firm and tender though not markedly so unless the sore throat is severe in which case the anterior cervical group may be very tender Usually only those on one side—most often the left—are involved at first but later on the enlargement becomes bilateral A few days after the glands have come up the spleen may be palpable but it is never notably enlarged Occasionally the liver also becomes moderately enlarged

The temperature continues elevated for a variable period most commonly for ten days or a fortnight The pyrexia may be continuous or remittent But unless the throat condition is severe the fever is by no means pronounced and the patient s general condition remains relatively very good The glands remain enlarged long after the temperature has come down and subside very slowly several months may

elapse before they return to normal Occasionally even when the temperature has apparently settled for good relapse occurs the mechanism of which is by no means clear

Sometimes in addition to or even in the absence of superficial glandular enlargement the deep glands are involved either the tracheo bronchial group or the mesenteric If the former there will be a harsh cough perhaps with bronchitis and radiological signs in the chest if the latter the tender glands may be felt by palpation and the suspicion of appendicitis aroused

Types —The foregoing is a brief account of the syndrome as commonly encountered along the lines first described by Pfeiffer But other types clinically different but with a similar basic pattern have since emerged In the *anginose* type the picture is dominated by the sore throat which may be intense with the formation of a false membrane very suggestive of diphtheria or alternatively it may be characterized by ulceration from which Vincent s organisms may be recovered In the *febrile* type the glandular enlargement is preceded by some weeks of pyrexia during the early stages of which a rash—macular maculo papular or even petechial may occur An *encephalitic* type characterized by dizziness diplopia and mental confusion has also been reported and also a *gastro intestinal* type with vomiting and diarrhœa as the leading signs

Complications —In a very small percentage of cases jaundice occurs perhaps due to hepatitis Hæmaturia may also occur Meningeal signs may imply the presence of a serous meningitis

Signs and Symptoms	Glandular Fever	Acute Lymphatic Leukæmia.
Fever	+ for 1–3 weeks	Usually + but Variable
Lymphadenopathy	+	+ +
Anæmia	–	+ to + +
Total White cells	+	– to + + +
Lymphocytes	+	+ + to + + +
Thrombocytopenia	–	+
Bone marrow	Glandular fever cells Halo cells Mononuclears up to 70%	Usually lymphoblasts but variable
General condition	Usually good	Poor Pallor weakness and hæmorrhages usual
Spleen	+	+
Paul Bunnell	Usually +	–

Diagnosis —This is far from easy in a sporadic case and can often be made only as the result of a lengthy process of exclusion The disease be it remembered is far from common indeed when one considers that sore throat is often prominent and that occasionally a rash occurs one might expect cases of glandular fever to crop up from time to time in infectious hospitals yet over a good many years one can recall very few The *anginose* form may be mistaken for diphtheria from which the absence of toxæmia the repeatedly negative swabs the blood changes and the axillary adenopathy should mark it off Alternatively it may be mistaken for simple Vincent s angina which may further complicate the issue by the presence of agranulocytosis Here again only the axillary and inguinal adenitis may provide a clue and only careful examination and extended observation can clarify this position The most likely sources of confusion with the ordinary form is an acute leukæmia especially acute lymphatic leukæmia (see table) or even Hodgkin s disease In this field the hæmatologist will play a decisive rôle as may he also in distinguishing those febrile forms in which a rash occurs from rubella measles the *enterica* or even secondary syphilis In rubella the transience of the febrile stage and the metamorphosis of the rash are characteristic Cases of glandular fever in which catarrh is pronounced may be confused with measles especially if conjunctivitis is present but careful search for Koplik s spots will prove fruitless The rash the fever and the enlarged spleen may suggest one of the *enterica* a suggestion to be dispelled by blood counts blood cultures and the serological reactions The

rash the sore throat and the adenopathy may also suggest secondary syphilis especially as the Wassermann may be weakly positive in many cases of glandular fever but the histories and courses of the two conditions are quite dissimilar and the blood changes of the latter when they appear are quite distinctive Incidentally sternal puncture may disclose these blood changes some days before they are discovered by the blood count

The Paul-Bunnell Reaction—Apart from the cytological changes *the serum in 75 per cent of the cases* of glandular fever is found to agglutinate the red cells of the sheep in dilutions of 1 in 64 or upwards sometimes to as high as 1 in 10 000 These heterophile' agglutinins may also be found in persons who have had horse serum but these horse serum agglutinins can be adsorbed by emulsions of guinea pig kidney which those of glandular fever are not The test is regarded as reliable

Treatment—Apart from convalescent serum no claims have been made for any particular substance or mode of therapy Of course in cases showing Vincent s ulceration penicillin or arsenic should be employed Arsenic indeed as N A B has recently been reported to have an inexplicable but markedly beneficial effect on glandular fever itself in cases where no Vincent s organisms were discoverable in the throat

Prognosis—Uniformly good

Prevention and Control of Outbreaks—As so little is known of the etiology preventive measures can be purely empirical at best It is reasonable to regard it as a droplet infection so that isolation, the wearing

of masks etc should be employed though indeed it may be spread by ingestion in which case appropriate measures will suggest themselves The disease is regarded as one of low infectivity as a rule so far as overt clinical signs are concerned but in one recent outbreak some 290 persons showed the typical blood changes and Paul Bunnell reaction whereas only 125 showed clinical signs of the disease a fact which suggests that infectivity may be much higher than has hitherto been supposed

Mumps

Mumps sometimes (unfortunately) designated **epidemic parotitis** is an acute infection which so far as we know is almost always characterized by enlargement of the *salivary* glands It is much more than a disease involving the parotids which seems to be a common conception of it

Cause —A virus cultivable with difficulty on chick embryo from a suspension made from infected (monkey) parotid gland The disease has been transmitted to monkeys by the intravenous injection of filtered saliva or mouthwashings from early cases

Pathology —The local lesion is usually situate in the nasopharynx which sometimes shows a sharp preliminary catarrh The virus may reach the salivary glands either directly via their ducts or through the blood Certainly on occasions it invades the blood stream to produce complications and sometimes it may do so to produce orchitis or encephalitis *without affecting the salivary glands at all* The parotids show

an interstitial and periglandular reaction whereas the testes, when involved show a parenchymatous inflammation consequently the former recover completely whereas atrophy of the testis is not uncommon The epididymus may also be involved The blood shows a considerable lymphocytosis but if orchitis occurs a polynucleosis The count is, however, very variable In mumps the serum lipase is usually normal but the serum amylase is increased in 95 per cent of cases, probably because of blockage of the parotid ducts

Incubation Period —Approximately three weeks

Segregation Period —A few days after swelling has subsided

Mode of Spread —Direct contact and droplet spray Carriers do not occur Outbreaks in schools and camps are common

Incidence —Mostly children from five to fifteen years and young adults Under three years it is almost unknown Males predominate One attack confers immunity

Varieties —One or both parotids are usually involved but the sublingual and submaxillaries may participate or the disease may be confined to them Submaxillary and sublingual mumps are often missed hence the occasional mysterious appearance of parotid mumps in a ward

Course —The stage of invasion is usually very short —twelve to twenty four hours but it sometimes lasts for two or three days with fever and a dry throat The angle of the jaw on the affected side next becomes tender and soon the parotid swelling appears most marked over the lower pole Swelling rapidly in

creases as does the periglandular reaction so that the hollow behind the angle of the jaw is obliterated, and if the submaxillary and sublingual glands participate

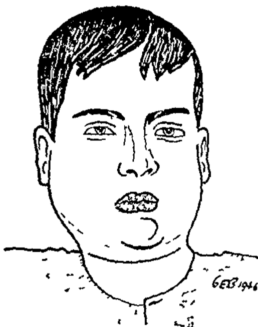


Fig 2 — Mumps—Compare with Frontispiece

a smooth tense curved swelling may run from the temperomandibular joint to the median raphe beneath the chin (see Fig 22) In a few days the opposing salivary glands may enlarge producing a ridiculous deformity the face assuming the appearance of a

an interstitial and periglandular reaction whereas the testes when involved show a parenchymatous inflammation, consequently the former recover completely whereas atrophy of the testis is not uncommon The epididymus may also be involved The blood shows a considerable lymphocytosis but if orchitis occurs a polynucleosis The count is however very variable In mumps the serum lipase is usually normal but the serum amylase is increased in 95 per cent of cases, probably because of blockage of the parotid ducts

Incubation Period —Approximately three weeks

Segregation Period —A few days after swelling has subsided

Mode of Spread —Direct contact and droplet spray Carriers do not occur Outbreaks in schools and camps are common

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bizarre complication ushered in by fever vomiting and prostration There is severe pain in the left hypochondrium and the overlying muscles are rigid though if not the enlarged tender gland may sometimes be palpable The urinary diastase index is raised and there may be acetonuria but very rarely glycosuria The stools may contain fat Rapid recovery is the rule **Otitis media or interna**—the latter perhaps followed by permanent deafness—may sometimes supervene

Encephalitis may also occur often in the form of a meningo-encephalitis Certainly abroad and especially in China signs of C N S involvement in mumps are much more common than in this country Changes in the C S F —increase in pressure and a pleocytosis showing counts of as many as 1000 cells per c c but with the sugar and protein normal are frequent and in a certain number of cases the clinical picture is that of an acute meningitis Indeed some authorities regard mumps as primarily an infection of the C N S and only secondarily of the salivary glands Such nervous signs are much commoner in adults than in children

Reversed or inverted mumps in which orchitis encephalitis meningism or pancreatitis may precede the parotitis or indeed constitute the sole manifestation of the infection may also very rarely occur

Diagnosis—It is surprising how often mumps is misdiagnosed The most frequent error is to confuse enlargement of the parotid and tonsillar *lymph* glands But those glands are usually movable and the swellings stop respectively above and below the jaw

hanging pear Opening of the mouth and mastication are difficult and painful though swallowing as such may be normal Each set of glands may continue to enlarge for two or three days, and may remain swollen for a week or longer before subsidence begins When fully developed the swellings are hard tense and tender and are not movable The orifices of Stenson s (parotid) duct (opposite the fifth upper tooth) and of Wharton s (submaxillary) duct (beside the frænum) may sometimes appear enlarged and congested Should the submaxillaries and sublinguals alone be involved hard tense swellings may be felt close to and often apparently integral with, the mandible

Fever usually slight abates as soon as the glands cease to enlarge Once begun the subsidence of any given gland is complete within about a week *whereupon it resumes its normal function* Suppuration is so rare as to cast doubt on the diagnosis and in the absence of complications convalescence is brief

Complications —Mumps is notable in that complications normally involve the sex glands **Orchitis** occurs in one in five males between puberty and senility *Roughly about the tenth day there is a* sharp resurgence of fever followed by painful swelling of one or both testicles The fever and swelling usually subside in a few days but unfortunately atrophy of the affected testicle not infrequently ensues As a corollary **oophoritis** is said to occur in women but clinical evidence of this is certainly very rare **Mastitis** does however occur *and may lead to a* similar atrophy **Acute pancreatitis** is a rare and

Prophylaxis—Convalscent serum or gamma globulin if available and given soon after exposure is of value

Control of Outbreaks—Segregate the culprit and quarantine for twenty eight days Bed isolation should be sufficient to check spread Exposed children may go to school for the first ten to fourteen days

The most disastrous error is to mistake the tonsillar adenitis and periadenitis ('bull neck') of hypertoxic diphtheria for mumps *The* **throat** *of every child diagnosed mumps* **must** *be examined* Other uncommon sources of confusion are *purulent parotitis* acute or chronic, enlargement due to *stone* in the duct parotid *tumours* *uveo parotitis* and *Mikulicz s* syndrome Sublingual or submaxillary mumps is very commonly mistaken for a lymphadenitis or a suppurative mandibular or dental condition indeed it is exceptional to find it spontaneously recognized Careful examination together with the history should allow of differentiation and a blood count showing the well marked lymphocytosis is often conclusive

Prognosis —As to life is invariably good but the risk of sterility in males is not negligible

Treatment —Little is usually required beyond a mouthwash and the relief of pain by sedatives and hot applications The patient may have to be fed on fluids through a tube Acid drinks such as the familiar lemonade which provoke salivation may cause exquisite pain and must be avoided The testis or breast should be supported if involved In regard to orchitis some observers believe that an acute hydrocele develops which by strangulation causes death of the testicle They advocate therefore that immediately signs of orchitis appear the tunica vaginalis should be drained when it is claimed the temperature will fall and the testicle will be saved Children may get up when the glands have subsided but males at or above puberty should stay in bed for a fortnight in the hope that a doubtful orchitis may possibly be avoided

enteric and typhus when these two diseases were erroneously regarded as one When they were separated enteric was re named typhoid or the typhus like fever To day confusion still exists to some extent because there are two main forms of typhus the *epidemic* (louse borne) type and the *murine* carried mostly by the rat flea *Epidemic* typhus is endemic in Eastern Europe and Russia and blazes up into devastating epidemics from time to time Doctors run special risks—in Ireland in an outbreak early in the eighteenth century it became known as the doctors murderer since some 40 per cent of all the practitioners in Dublin died of it *Murine* typhus on the other hand does not it would appear become epidemic but is endemic in many parts of the world Allied infections occur under such names as Rocky Mountain Spotted Fever and Brill s Disease in the U S A Bush Typhus in Malaya Tsutsugamushi in Japan Q Fever in Australia and so on These diseases are carried by ticks mites and other insects

Both *epidemic* and *murine* typhus are caused by distinct but closely related forms of *Rickettsia* agents which it is now generally agreed are probably viruses Points of difference are that the virus of murine typhus (*Rickettsia Mooseri*) injected into the white mouse causes the death of the animal whereas that of epidemic typhus (*Rickettsia prowazeki*) leaves it almost unaffected Again the murine virus injected into the *tunica vaginalis* of the guinea pig gives rise to a severe reaction in which cells full of Rickettsiæ—the Mooser cells—are to be seen whereas epidemic virus produces only a little local induration though-

SECTION XII

TYPHUS FEVER AND INFLUENZA

Typhus Fever

DEFINITION —An acute infectious disease characterized by intense fever a rash extreme prostration and a high mortality

There is no disease that better illustrates the influence exercised on the public health by the standard of living in general, and by personal hygiene in particular than typhus Formerly so prevalent in this country especially in our jails and prisons that the judges carried bouquets in court in the pious hope of warding off infection it is now so rare that when the first edition of this book was prepared some years ago and found to exceed the stipulated length a section which had been written on typhus was discarded without hesitation since as far as I could find out no case had been seen in a London infectious hospital for a generation Indeed until the final stages of the recent war when a few cases were imported from the Continent the British Isles had remained singularly free from the disease if we except an occasional case *brought into a seaport town or reported intermittently* from the West of Ireland

Aetiology —Confusion in nomenclature seems to be especially associated with typhus The word which appropriately enough implies a confused or clouded state of the mind was formerly applied to both

'unsalted' community than in one in which the disease is endemic had any cases been imported into this country during the blitz periods when shelter life led to overcrowding and an increase in louse infestation the results might have been disastrous In countries where the disease is endemic the lowest incidence is from June to October while the peak occurs in the March–May three months a period when not only is the weather still cold thus fostering overcrowding indoors but the diet is likely to be deficient in vitamins while the effect of the summer and autumn vitamin intake has largely worn off

Pathology—There is a generalized congestion of the liver spleen etc common to most acute infections The only lesions peculiar to typhus are to be found in the blood vessels where there is a patchy necrosis of the intima of the small arterioles which sometimes extends to the muscular layer often at the same time there is a perivascular infiltration of lymphocytes plasma cells mononuclears and polynuclears around the affected part of the vessel The Rickettsiæ can be demonstrated in the infiltrating cells These lesions are to be found not only in the vessels involved in the rash but in the brain the heart muscle etc The heart muscle may show patches of necrosis The cerebro spinal fluid shows a pleocytosis The *blood* changes are inconstant There may be a leucopenia at the beginning but subsequently there is a polynucleosis The red cells are normal or slightly increased

Incubation Period—This averages eleven to twelve days though a minimum of five and a maximum of twenty has been recorded

extensive nodular lesions develop in the brain Finally whereas the host of the murine virus is the rat or other rodent the host of the epidemic virus as far as we know is man alone

The virus appears to circulate in the blood of the typhus patient during the febrile period of the disease When a louse bites such a patient (and thereby infects itself) the Rickettsia lodge in the cells lining the louse s gut where they multiply In about five days they begin to be shed into the lumen of the gut and thus to be excreted in the fæces If the louse be now transferred to an uninfected person and bite* him the infection is not conveyed as in plague during the act of biting but by the subsequent inoculation with the Rickettsia-laden louse fæces of the bite or of some other abrasion as the result of scratching The louse concerned is the body or head louse which succumbs to the infection itself in a few weeks

Epidemiology—*It is obvious that circumstances* which produce overcrowding and discourage personal hygiene so that lice flourish and can be readily trans*ferred from one person to another provide the essential* conditions for an epidemic of typhus A second and most important factor is malnutrition War provides both these conditions in superlative degree and it is therefore either during or following a war that the most notable epidemics have occurred Recent history has once again underlined this malignant association for typhus became rampant in the concentration camps and in many prisoner of war camps in Germany and Eastern Europe generally

Naturally typhus will spread more rapidly in an

* The louse does not bite but rather pierces the skin

shakes and there may be tremors of the tongue Soon there is an intolerable sensation of complete exhaustion The tongue indeed may become so weak that it cannot be protruded it is heavily coated As might be expected with such acute fever dehydration is marked thirst is intense constipation is usual and the urine is scanty and highly coloured It may contain albumin and the diazo reaction (see page 172) is usually demonstrable

The temperature rises quickly to a maximum of 104 to 105 F by the second or third evening and remains elevated without remissions until defervescence occurs The pulse is soft but not at first greatly accelerated though later in unfavourable cases it may rise to 140 or more The blood pressure falls the systolic sometimes to less than 100 mm *Hg* the respirations also become rapid leading perhaps to a belief that pneumonia is impending The spleen may become palpable but is never considerably enlarged

The first suggestive diagnostic sign in an unsuspected case is the rash This usually appears about the fourth or fifth day and is first seen on the abdomen or about the axilla whence it spreads all over the body with the exception of the face It may be seen on the palms and soles but it is always heaviest on the trunk Classically it consists of three elements two of which are common The first to appear are small pink maculo papules smaller and flatter than the rose spots of enteric and which likewise disappear on pressure but only during the first few days By that time they have lost their pink appearance and have become a dirty brown In the centre of these spots petechiæ may appear and can readily be produced on the arm

Signs, Symptoms and Course — It takes some time to become accustomed to the diagnostic short cuts which are necessary in North China No Westerner would consider the diagnosis of typhus fever as a first guess in a patient who comes down with acute fever, severe headache and some leucopenia during the winter or the spring but this is necessary in Peiping One understands better why the typhus epidemic in the prison camps in Germany during the World War of 1914 was mistaken for several months for an *influenza epidemic* *Especially during the first four* to five days of the disease it may be extremely difficult to differentiate between typhus and influenza

Thus an American physician practising in Northern China In the louse borne fever of Europe the picture is similar if intensified The patient who may have felt off colour during the last few days of the incubation period takes to his bed with shivers severe headache and pains in the back and limbs There is usually bronchitis and conjunctivitis but very soon the stage of nervous excitement may supervene and the patient become acutely delirious and attempt to escape In a few days however the delirium passes off and the patient relapses into the condition so well described by Murchison The expression at first betokens languor and weakness but it soon becomes dull heavy and stupid sleep is disturbed by painful dreams and sudden starts and after three to four nights there is talking in the sleep with slight delirium between sleeping and waking When awake the patient is still conscious though perhaps confused in memory and intellect With all this there is early and increasing muscular prostration The hand

lysis or even by a dramatic crisis He is of course grossly enfeebled and may yet collapse Cautious and prolonged convalescence is necessary for full return to health

Varieties —Typhus may vary from a mild disturbance of health in districts where it is endemic to a terrible disease that strikes and kills in a few days *Typhus siderans* (Blasting Typhus) is a name given to such a form Rarely extensive hæmorrhages characterise *hæmorrhagic typhus* but this variety is rare

Complications —These fall mainly into two groups—those resulting from the failing circulation and those resulting from infection of the lungs Respiratory infection is especially to be feared because of the foulness of the mouth unless it receives constant attention Septic parotitis is not uncommon the gland may even go gangrenous Some bronchitis seems to occur in most sharp cases and bronchopneumonia is not infrequent On the other hand hypostatic pneumonia is common among older patients Enteritis may also occur perhaps with melæna Femoral thrombosis is more frequent than in enteric Bed sores become gangrenous and œdema and gangrene of the fingers and toes and even of the nose may reveal how seriously the circulation has become impeded The involvement of the nervous system may as noted result in temporary deafness and even transient mental changes—mania melancholia etc may also ensue

Mortality —Few diseases exhibit such a wide range of mortality as typhus Age nutritional state and previous experience of the disease all play their part

by means of a tourniquet These maculo papules may continue to come out for two to three days but after that no more appear they sometimes congregate in blotchy patches They persist for about ten days though the petechiæ endure for longer

The third, and rare element of the rash is the sub cuticular mottling This looks as though the rash had come out in larger and irregular patches beneath *the cuticle, giving to the skin a reddish brown mottled* appearance The complete picture is sometimes described as the mulberry rash' of typhus largely because of the purple of the petechiæ In a small number of cases the whole rash is abortive while in very severe ones petechiæ may be so abundant as to dominate the picture

As he passes into the middle of the second week the patient likewise passes into the typhoid state He lies completely prostrated and heedless, his face dusky and stupid his eyes injected and half open with the pupils narrowed to pin points (ferret eyes) his tongue shrivelled black and tremulous his teeth gums and lips encrusted with sordes The nose and extremities may become cyanosed As he lies picking at the bedclothes with tremulous hands and muttering to himself (*coma vigil*) he exhales a dull mousy odour He may be—or appear to be—completely deaf The pulse rate steadily increases and the tension falls and he is now incontinent He may quieten fall into a deeper coma and die usually towards the end of the second week Alternatively about the same time he may pass gradually into a genuine sleep from which he awakes with a moistening mouth and a clearing mind to make a recovery, while the temperature falls by a rapid

than that of enteric prostration is earlier and more profound and enteral signs are exceptional

The Weil-Felix Reaction —Certain O (deflagellated) strains of *B proteus*—the X19 X2 and K seem to share a common antigen with the Rickettsia In epidemic or murine typhus after the end of the first week the patient s serum should agglutinate the X19 group in a rising titre of 1/200 or over and this may attain to 1/10 000 in convalescence At least 75 per cent of cases show this phenomenon which is consequently of great value in diagnosis For more exact differentiation of the Rickettsiæ involved complement fixation tests with Rickettsiæ cultivated on chick embryo are required

Treatment and Management —Before admission to the ward the patient should be deloused Personnel engaged in delousing and in the wards should not be more than thirty years of age and should wear protective clothing of which the essentials are that it should be made in one or at most two pieces cover the whole body fasten tightly at the neck wrists and ankles by means of strong elastic and be composed of some light-coloured material against which the lice can easily be seen They should also wear rubber gloves and gum boots The patient should be stripped in a special bathroom his clothes the ambulance blankets etc should be placed in a bag containing crude naphthalene which is then placed in a covered bin This kills the lice Six hours later the bag is removed and placed in a steam disinfector where the Rickettsiæ are killed The patient is shaved except for the head which is fine-combed and if infested treated with lethane and covered with a cap

age playing the most important part Thus in one series the death rate was less than 2 per cent for the age group ten to fifteen years but 60 per cent for the age group fifty to sixty years Again it was 7 per cent among the young Russian prisoners in camps during the 1914–18 war but 58 3 per cent among the older German doctors who were attending them In Riga in the 1918–20 epidemic the average mortality was 13 6 per cent with no deaths in children under ten years

Diagnosis —During an epidemic diagnosis is not difficult It is the occasional sporadic case that may cause so much confusion Again much depends on the mode of onset of the disease At the outset influenza may be strongly suggested, or cerebro-spinal fever in cases where the nervous signs are marked Smallpox with a prodromal macular rash may also be suspected Where there is early stupor the case may appear to be one of encephalitis Time and the appearance of the rash will quickly clear up confusion with influenza or encephalitis and smallpox will rapidly declare itself Clinically the differentiation from enteric may offer great difficulty and in former days much stress was laid on the differential diagnosis on clinical grounds such as the disappearance of the enteric rash on pressure but nowadays blood culture and the agglutination reactions in enteric and the Weil Felix reaction in typhus should soon provide conclusive evidence Clinically the rashes of the *enterica* are more papular disappear on pressure and appear in crops Those of the para typhoids may invade the face which the rash of typhus never does The onset and course of typhus is much more abrupt

used by the U S Army to deal with the local Rickett sial infections during the recent war and with great success) Finally there is Blanc s vaccine consisting of *living* murine virus which is also said to be effective Clearly the use of live virus possesses dangers of its own for if it should appear in the blood in a louse ridden community it might spread the disease The British Army in the recent war checked incipient epidemics by drastic and thorough delousing forcing DDT in powder form under the garments of all those at risk and treating the heads (cropped if necessary) with lethane

Influenza

Influenza is an acute and very highly infectious disease sometimes of protean symptomatology but usually marked by acute upper respiratory catarrh

Epidemiology —The epidemiology of influenza is a baffling subject about which very little is so far known with certainty Epidemics occur about every couple of years in this country and indeed all over Europe and in the United States Between times the disease seems to disappear and whence the fresh cases from which the next epidemic arises derive their infection is a matter so far unexplained As men tioned on page 49 the agent of swine influenza a closely related disease is believed by Shope and others to spend the inter epidemic periods in the lungworm which infests the pig When the pig dies the lung worm is again ingested by other pigs and if at this stage the cold weather suddenly makes its appearance an epidemic of swine influenza may be expected to

He is then bathed and dressed in hospital nightwear which may with advantage be impregnated with DDT In case any nits may have escaped the head should be treated again with lethane in five days' time

As no diseases exemplify the febrile state more completely than do typhus and typhoid so none offer more scope for the classical treatment of this condition as described on pp 175 178 Of specific treatment there is little of much value Convalescent serum has been warmly advocated and seems to have given good results but it is unlikely to be available in this country Administration of the B group vitamins as a whole and especially of para aminobenzoic acid has recently received very high praise

In the stage of excitement a lumbar puncture is often helpful while in the stage of collapse a variety of stimulants ranging from brandy by mouth to the latest refinements of the manufacturing chemist given intravenously in glucose saline may be exceedingly useful

Prevention —Since if there are no lice there can be no typhus in these islands the best and most obvious preventive measure is the steady improvement in personal hygiene In countries in which either epidemic or murine typhus occurs vaccination is to be recommended It will not it would seem always prevent the disease but it will diminish its intensity Several vaccines are available—Weigel s consisting of the macerated bodies of lice which have been inoculated per anum with Rickettsia and now contain killed virus and Cox s consisting of formalinized Rickettsial suspensions grown on chick embryo (Large numbers of vaccines composed of the virus thus cultivated were

Varieties—The disease may be classified according as the predominant signs are *respiratory nervous* or *gastro intestinal*. The respiratory is the ordinary type. Both the nervous and gastro intestinal types are rare; indeed some clinicians believe that a gastro intestinal type *pur sang* does not exist.

Signs Symptoms and Course—The onset is sudden with fever shivering headache a dry or even definitely sore throat and pains in the limbs and back. In the common respiratory type there is usually in addition coryza and mild bronchitis with a dry cough. Sometimes there may be labial herpes. Headache too is usual though different epidemics often display slight differences in symptomatology. For instance in the 1918 (summer) outbreak pain on looking upwards was a characteristic symptom. At the same time there are the usual concomitants of the febrile state anorexia perhaps nausea and vomiting and it may be some abdominal pain.

In the uncomplicated disease these signs and symptoms may persist for two or three days following which the temperature falls to normal and the patient feels very well and is usually anxious to get up at once. On getting up however he may find himself to his surprise quite disproportionately enfeebled by such an apparently trivial illness. Rapid fatigue and palpitation on exertion are prominent symptoms and it may be a fortnight or a month before these symptoms abate and full return to normal health is secured.

In the nervous type of the disease dizziness may be prominent and the patient may be restless and unable to sleep. Occasionally he may be delirious. Since

appear No similar resting place for the virus of human influenza has however so far been discovered

Influenza has appeared many times in history and a prime characteristic is the speed with which it spreads through a community It is of course a droplet infection The memory of the great epidemic of influenza during 1918–19 is still fresh and the mortality by which it was marked remains unique in the annals of modern epidemiology This outbreak was characterized by three successive waves of which the second was the most lethal

Although as noted epidemics appear fairly regularly the epidemiology of the disease as already noted is most capricious and especially in recent years it has often failed to materialize when confidently expected

Cause—Influenza is caused by a virus of which two strains the A and B have so far been indentified Other strains may exist The virus has been cultivated with great ease on chick embryo and vaccines have been prepared from these cultures Virus A would appear to be the more virulent of the two besides surpassing its rival in invasiveness and in infectivity Other types may yet be identified

Incidence—All ages Repeated attacks are common—in fact the rule In the 1918 epidemic mortality was highest among young adults but in normal epidemics the aged suffer most severely

Incubation Period—*One to three days at most*

Segregation Period—The disease is most infectious during the early catarrhal stage By the time the patient is fit to get up infectivity would appear to have disappeared

Varieties —The disease may be classified according as the predominant signs are *respiratory nervous* or *gastro intestinal* The respiratory is the ordinary type Both the nervous and gastro intestinal types are rare indeed some clinicians believe that a gastro intestinal type *pur sang* does not exist

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similar symptoms may occur in the febrile state due to other infectious agents it is doubtful if, from these alone we are entitled to assume that the virus has made a specific attack on the nervous system rather than that nervous symptoms of an ordinary attack are more than usually prominent

This last remark would also seem to apply to the gastro intestinal type of the disease In some persons nausea especially may be extremely troublesome and it may be associated with vomiting pain in the abdomen and diarrhœa Since the virus normally abounds in the naso pharynx and must be continuously swallowed it is perhaps not surprising that gastro-intestinal symptoms should sometimes be experienced and that occasionally they should be unduly assertive

Complications —Influenza itself is a trivial disease but its complications may be most formidable The commonest are those which arise under the ægis of the catarrh in the upper respiratory tract Of these bronchitis is much the commonest but broncho-pneumonia has achieved the most sinister reputation In the 1918 epidemic the familiar violet cyanosis might appear as early as the second or third day to announce that bronchopneumonia was established and that a fatality might be expected within a further twenty four or forty-eight hours When the patient survived pleurisy and empyema might follow as in any broncho pneumonia or even lung abscess though this was less common The maxillary antrum any of the sinuses or the middle ear may participate in the inflammation and otitis media may easily lead to mastoiditis All these complications are of course due to secondary

organisms notably the *hæmophilus influenzæ* in the cyanotic bronchopneumonia and the strepto or pneumococcus in the more ordinary pneumonia or in the sinuses or middle ears organisms for which the virus by its preliminary work has rendered the path of invasion an easy one

Complications due to the virus itself are however by no means lacking The first of these is *carditis* It seems clear that in a large percentage of cases the virus actually invades the myocardium where in a few instances it has actually been demonstrated This would account for the pronounced weakness so commonly experienced in convalescence Should the virus invade the conducting bundle disorders of the cardiac rhythm will ensue and can sometimes be detected in the pulse and more frequently by the electrocardiogram. Complete heart block may thus result though this is very rare

The second complication directly due to the virus is *encephalitis* Overt encephalitis with diplopia drowsiness and perhaps nuclear cranial nerve palsy is not common but in quite a number of cases severe mental depression sometimes so pronounced as to lead to suicide follows influenza It should also be mentioned that a number of cases demonstrating the post encephalitis lethargica syndrome have been noted which seemed to date from an attack of influenza though whether correctly or incorrectly diagnosed it is not, of course possible to say

Diagnosis.—The diagnosis of influenza is not difficult so characteristic and common is the condition the danger is rather that during an epidemic anything remotely resembling the disease tends to be diagnosed

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similar symptoms may occur in the febrile state due to other infectious agents it is doubtful if, from these alone we are entitled to assume that the virus has made a specific attack on the nervous system rather than that nervous symptoms of an ordinary attack are more than usually prominent

This last remark would also seem to apply to the gastro intestinal type of the disease In some persons nausea especially may be extremely troublesome and it may be associated with vomiting pain in the abdomen and diarrhœa Since the virus normally abounds in the naso pharynx and must be con tinuously swallowed it is perhaps not surprising that gastro intestinal symptoms should sometimes be experienced and that occasionally they should be unduly assertive

Complications —Influenza itself is a trivial disease but its complications may be most formidable The commonest are those which arise under the ægis of the catarrh in the upper respiratory tract Of these bronchitis is much the commonest but broncho pneumonia has achieved the most sinister reputation In the 1918 epidemic the familiar violet cyanosis might appear as early as the second or third day to announce that bronchopneumonia was established and that a fatality might be expected within a further twenty four or forty eight hours When the patient survived pleurisy and empyema might follow as in any broncho pneumonia or even lung abscess though this was less common The maxillary antrum any of the sinuses or the middle ear may participate in the inflammation and otitis media may easily lead to mastoiditis All these complications are of course due to secondary

organisms notably the *hæmophilus influenzæ* in the cyanotic bronchopneumonia and the strepto or pneumococcus in the more ordinary pneumonia or in the sinuses or middle ears organisms for which the virus by its preliminary work has rendered the path of invasion an easy one

Complications due to the virus itself are however by no means lacking The first of these is *carditis* It seems clear that in a large percentage of cases the virus actually invades the myocardium where in a few instances it has actually been demonstrated This would account for the pronounced weakness so commonly experienced in convalescence Should the virus invade the conducting bundle disorders of the cardiac rhythm will ensue and can sometimes be detected in the pulse and more frequently by the electrocardiogram Complete heart block may thus result though this is very rare

The second complication directly due to the virus is *encephalitis* Overt encephalitis with diplopia drowsiness and perhaps nuclear cranial nerve palsy is not common but in quite a number of cases severe mental depression sometimes so pronounced as to lead to suicide follows influenza It should also be mentioned that a number of cases demonstrating the post encephalitis lethargica syndrome have been noted which seemed to date from an attack of influenza though whether correctly or incorrectly diagnosed it is not of course possible to say

Diagnosis—The diagnosis of influenza is not difficult so characteristic and common is the condition the danger is rather that during an epidemic anything remotely resembling the disease tends to be diagnosed

as influenza Any febrile condition before it declares itself may be mistaken for it and to give the differential diagnosis in detail is quite outside the bounds of space at our disposal The point to remember is that during an epidemic most diagnoses of influenza will be correct but that a certain proportion will be mis taken each case therefore should be followed to its conclusion before the diagnosis is finally and con fidently confirmed

Treatment and Management—The first and most essential measure in influenza is to put the patient to bed this is usually easy The second is to keep him there—and this is usually most difficult Almost everybody gets up too soon after influenza The patient feels quite well at rest and rarely suspects that he may have an active—if temporary—myo carditis from which he may recover much more readily if he is content to remain where he is

For the rest treatment is purely symptomatic Aspirin or A P C for the headache hot lemon drinks or whisky toddy (if available) gargles and so on are prescribed as occasion arises A Dover s powder is a favourite remedy to induce sleep In sharp cases or where complications seem likely the sulphonamides may be given prophylactically but as always with caution Where possible the patient is isolated and treated as the highly infectious individual that he is but it is difficult to persuade the public to take in fluenza as seriously as its sinister record deserves

Once the fever has abated the patient may have an unrestricted diet In mild cases he should stay in bed for at least three days after the temperature has subsided in more severe cases a week should be

insisted upon He may be warned that he may find himself unduly depressed for a week or two and if so to remember that this is part of the pattern of the disease from which he will in due course recover completely

Prophylaxis—As stated earlier on the virus cultivated on chick embryo has been employed by many workers to produce vaccines but so far the results of inoculation have been disappointing In Australia the vaccine has been introduced intravenously and antibodies have been demonstrable in the blood following this procedure Elsewhere the vaccine has been administered subcutaneously in the ordinary manner Unfortunately the results so far have been disappointing a couple of months immunity at best resulting in man This accords more or less with clinical experience since repeated attacks of influenza are commonly experienced by the vast majority of the population It is believed by some that the explanation of this phenomenon may lie in the fact that whereas in those diseases where a permanent immunity results the virus remains permanently in some of the body cells in influenza this does not happen hence the transience of the immunity

As long as we insist in herding together in towns and cities in trams buses trains theatres and cinemas droplet infections of the respiratory tract can scarcely be avoided The only certain preventive of influenza is to shun sources of infection whenever we can detect them During epidemics we can wear masks when attending our patients remembering that several layers of gauze are required to give reasonable protection and that such masks must frequently be changed

The value of prophylactic gargling however highly advertized the substance employed may be is doubtful and even if the nose were adequately irrigated at the same time the procedure would still remain questionable

SECTION XIII

MISCELLANEOUS

INFECTIOUS DISEASE AND THE LAW

THE occurrence of certain of the acute infectious diseases is compulsorily notifiable to the Medical Officer of Health in theory by the head of the family and the medical practitioner attending the patient but in practice invariably by the latter He is to be provided with free forms for certification and paid 2s 6d for his trouble unless he notifies in his capacity as medical officer of any public body or institution in which case he is entitled to only 1s He is liable to a fine of 40s if he fails to notify unless he can satisfy the Court that he had reason to believe that the notice had already been sent

The notifiable diseases (in England and Wales) are smallpox cholera diphtheria membranous croup erysipelas scarlet fever typhus typhoid enteric or relapsing fever plague cerebro spinal meningitis acute poliomyelitis tuberculosis ophthalmia neonatorum encephalitis lethargica malaria dysentery (bacillary or amœbic) acute primary pneumonia acute influenzal pneumonia puerperal pyrexia measles and whooping cough Scabies is notifiable in London Anthrax must be notified to the Chief Inspector of Factories

This list may be enlarged as circumstances require by the Minister of Health or by any Local Authority

provided that they first obtain the approval of the Minister advertise it in the local press and notify the local practitioners Thus should an epidemic of smallpox occur, chickenpox would probably be made notifiable lest a misdiagnosis result in a case of small pox escaping observation

The first thing therefore that the new practitioner should do is to ascertain the whereabouts of the local *Health Authority*, and what diseases are *notifiable* in the locality and obtain a supply of notification forms (free) from the Authority When a case occurs he may notify the Authority by telephone leave one certificate for the ambulance nurse and post one on to the M O H (Unless he supplies a certificate he will not be paid) The certificate which can in emergency, be written on any piece of paper should run as follows

I certify that A—— B—— of———— is suffering from ———— If he wishes he may add ' and should be or is fit to be—removed to hospital ' Note that to say is probably suffering from ——" is not a valid certificate nor can '*contacts* be legally removed to hospital Furthermore and more im portant the law in England and Wales gives no power to segregate or detain *carriers* The patient must be *suffering from* the disease The M O H can only debar carriers of the enterica or dysenteries from employment in any place where food or drink is handled In Scotland a carrier may be segregated for three months on the certificate of the M O H and another practitioner that he is a danger to others, by reason of the probability of his spreading infectious disease This certificate is renewable though the carrier may appeal to the Board of Health -

Any person knowing that he is suffering from a notifiable disease who exposes others to the risk of infection by reason of his presence in any street theatre club shop etc is guilty of an offence, as is anyone who being in charge of him permits him to do so

The certified person must use no public vehicle nor may he hire a cab without disclosure and payment of the cost of disinfection A practitioner therefore who sends such a patient to hospital by tram or bus may be subject to a penalty The infected person may not engage in any employment in which there is risk of infecting others nor may a child attend school until the M O H certifies that he may safely do so

The certificate enables the patient to be removed to an infectious hospital though he need not necessarily be so removed if the M O H considers that he can be nursed at home without serious risk of infection to others Should the M O H come to the contrary conclusion and the patient resist removal an order can be obtained from a Justice of the Peace to effect this forcibly In the same way a patient in hospital can be forcibly detained though such stringencies are rarely resorted to or required

As with the patient so with his effects He may not borrow from nor return books to a public library nor may fomites be sent to the laundry nor even placed in the dustbin without disinfection The premises must be disinfected subsequently and may not be let otherwise The local authorities usually do it free Should the patient die the M O H or other practitioner may direct that the body be removed from the house or hospital direct to the mortuary for burial nobody shall be permitted to approach it unnecessarily

and the holding of a wake shall be visited with a penalty

These regulations were originally made to control the spread of smallpox typhus etc Formerly the local authority could compulsorily vaccinate contacts of smallpox but nowadays they may only offer to do it free of charge the M O H may however, forcibly delouse contacts of typhus

Infectious hospitals were *first* introduced *not to treat* but to lessen the incidence of infectious disease patients were therefore segregated in them not primarily in their own interests but in the interests of the community From this follows one important point—treatment in such hospitals is free of charge in contrast to general hospitals maintained by local authorities where some of if not all the cost of maintenance *must* be recovered from the patient or his family In future of course it will be free

Mechanical Artificial Respiration

Artificial respiration by mechanical means has achieved much prominence of late especially on account of the publicity it has received in the press Respirators may be divided into two types (*a*) **vacuum** respirators and (*b*) **pressure** respirators Examples of (*a*) are the *Drinker Both* and *cuirass* machines The two former are coffin shaped metal or wooden boxes in which all but the patient s head is enclosed At one end is the neck aperture which can be so adjusted by means of shaped metal plates and a rubber collar as to provide an airtight joint

The head lies on a padded rest At the opposite end is the opening of a flexible pipe which connects with an electrically driven bellows roughly of a concertina

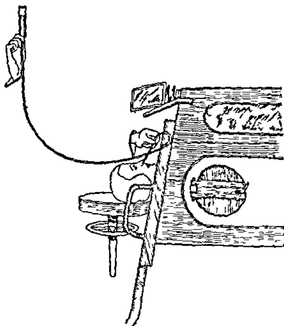


Fig °3—Child in Drinker Resp rator A nasal feed is being a lr in stered Pref rably the ch ld should lie on her s d

type When the bellows expands air is sucked from the respirator creating a vacuum As a result the chest wall expands carrying with it the lungs into which air at atmospheric pressure is drawn When the bellows contracts air at atmospheric pressure is

admitted to the respirator the chest wall recoils and thus accomplishes expiration The depth and frequency of the vacuum can be varied normally twenty double strokes per minute with a vacuum pressure equal to 25 cm of water are employed The respirator can be illuminated warmed and tilted Ports are provided carrying rubber sleeves so that the patient may be attended to without loss of vacuum pressure In the cuirass type similar principles are applied to an aluminium jacket made in two halves designed to enclose the thorax alone leaving the limbs free Airtight joints for the arms neck and waist are provided

Type (*b*) is represented by the *Bragg Paul* pulsator Here a flattened tubular rubber belt is secured around the patient s thorax and alternately inflated and deflated by a bellows On inflation the chest wall is compressed expelling the air in the lungs on deflation the chest wall re expands resulting in inspiration

Both types may be of great value not only in drowning gas poisoning and other forms of asphyxia but in such conditions as diphtheria poliomyelitis polyneuritis etc where the muscles of respiration are paralysed In general the box type vacuum machines are much the more efficient but the Bragg Paul shares with the cuirass " the advantage that the patient s limbs are free if he is able to use them a point of much psychological importance The former however, requires a good resilient chest wall (with unimpaired intercostals) to give of its best

In such cases of respiratory paralysis as show central involvement, the patient may fail to synchronize his breathing with the machine These offer a bad prog

nosis Further should the patient be confined to the machine for a long period pneumonia may develop, and bed sores occur though in the absence of a fatal infection he may be kept alive indefinitely Unfortunately the position of his head (see Fig 23) almost invites infection from anybody who talks to him and both nurses and visitors should be cautioned *not* to breathe into his upturned face and *never* to approach him with a cold or sore throat Most of such patients die of a pulmonary infection all those in attendance or visiting should wear efficient masks

Some Common Mistakes

If to err be human then no practising physician can have his humanity called in question To make mistakes is the common lot and the practitioner who believes himself infallible has never learnt anything None the less if we are forewarned of the pitfalls that await us we may minimize our errors to that end the following notes are included

(1) In babies *under six months* diagnose scarlet fever rubella or mumps with the greatest caution It is unlikely that they ever occur Measles is also very rare only occurring in babies whose mothers have not had the disease

(2) Remember the incubation period The figures given are the extreme limits A child *cannot* develop chickenpox say only three days after exposure

(3) Remember the segregation period A child cannot give rise to measles in others a fortnight after his own rash Not every child that whoops is infectious

Incubation and Segregation Periods

Disease	*Incubation P*	*Segregation P*
Scarlet F	2–4 days	Until all discharges ceased
Diphtheria	2–4 ,,	Until two weekly cultures negative
Erysipelas	1–4	None enforced
Puerperal F	1–14 ,,	Until all discharges ceased
Cerebro spinal F	2–4	None enforced
Poliomyelitis	4–21 ,,	One week from paralysis
Dysentery	1–7	Until three weekly stools "negative"
Pertussis	7–14	Four weeks from the first whoop
Enteric	10–14	Until stools and urine 'negative"
Smallpox	10–14	Until all seeds separated
Chickenpox	10–21	Until all crusts separated
Measles	10–21	One week from true rash
Rubella (see p 276)	14–21	One week from true rash
Mumps	14–28	On subsidence of glands

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